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MASTOIDITIS COMPLICATED BY UNUSUAL NEUROLOGIC MANIFESTATIONS. A CASE FOR DIAGNOSIS.*

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R. S., female, white, age 20 years, was referred to me by Dr. Jacob Gutman on Dec. 19, 1930. She complained of pain in the left ear of three days duration accompanied by a spontaneous discharge, dizziness, vomiting, tenderness over the left mastoid region and a temperature of 102°.

This patient was in the midst of an investigation for another ailment, thought to be a cystic degeneration of the pituitary, when the study of her case was suddenly interrupted by an acute ear infection. The record obtained by me from her attending physician is extremely interesting and important. I consider it a most valuable contribution to the report of the case. The following is an abstract of the record as of Dec. 15, 1930:

Family History: Father and mother living and well. Five sisters and three brothers in good health.

Habits: Normal in every way.

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Past History: In childhood: measles, scarlet fever, diphtheria and tonsillitis. Began to menstruate at 10½, regularly but rather scanty. Patient is single, not keeping company. Last menstrual period, Dec. 7, 1930, within the normal. Has always been in good health until about one and one-half years ago, when she began to increase abnormally in weight (about 50 pounds within a few months). About a year ago she experienced a sensation of numbness in the lower half of the body. Soon after, she felt a weakness and tingling sensation in the left lower and upper extremities, which is still persistent. About seven months ago the patient had a left facial palsy, which lasted for about two weeks. This was followed by diplopia, which also disappeared. She is subject to colds, but no ear trouble.

Present Complaint: For the past three weeks the patient has been suffering from nausea, and attacks of retching and vomiting, especially in the morning. She has been suffering with fits of depression, diminution of mental acuity and spells of dizziness, but no headaches. She sees well, hears well, has had no loss in weight, has no rheumatic symptoms, but her gait is somewhat unsteady, and with some stumbling.

Physical Examination: Height 4 feet 9½ inches, weight 164 pounds, pituitothyrotrope constitution, lax and heavy abdomen, flat feet, cold and blue hands, left eye drowsy and somewhat deeper than the right, many fillings in the teeth, tongue coated, thyroid slightly enlarged, lungs and heart negative. *Blood Pressure:* Systolic, 110; diastolic, 80; pulse rate, 66. *Gastric Analysis:* Normal. *Blood count* as of Dec. 16 shows R. B. C., 4,860,000, with hemoglobin, 94 Sahle units; leukocytes, 8,600, with 58 per cent polys.; platelets, normal; coagulation, normal. *Complement fixation* tests for syphilis (Noguchi, Kahn and Meinicke), negative. *Urine Analysis:* Loaded with am. phosphates, otherwise negative. *Spinal Fluid:* 8 c.c. pressure, 26 m.m.; Hg., clear; cell count, 8 per c.m.m.; dextrose, 86 m.gm. per 100 c.c.; protein, 64 m.gm. per 100 c.c. *Wasserman:* Negative. *Basal metabolism* rate, minus 11. *Dextrose tolerance test* faintly positive two hours after administration of dextrose; negative after the first, third and fourth hours.

Radiologic examination as of Dec. 18: Stereoroentgenography of the head shows a perfectly normal calverium, with the exception of a moderate projection of the posterior occipital region above the lateral sinuses, which are very large and deep. There are evidences of some intracranial pressure in the occipital region, as shown by finger markings and depressions. There is a slight thickening of the

tentorium cerebelli in the region of the sagital suture. The sella turcica is of average size. The anteroposterior diameter is about 12 m.m., the depth about 8 m.m. The clinoid processes are delicate, apparently normal. The floor of the sella well outlined. The sphenoidal sinuses anteriorly, clear; posteriorly, osseous.

Neurologic analysis as of Dec. 16: Gait and station, sways to the left. She walks with a tendency to the left hemiplegic gait, no tremors, no spasms, no deformities; neither hypo nor myotonia. The left upper extremity shows some inco-ordination. All sensory functions are intact, no astereognosis, no spinal rigidity, no tenderness. All cranial nerves are of normal function.

Reflexes: Knee: right, 3+; left, 4+; Achilles, 2+; plantar, 0; Babinski: right 1+(?); left, 1+. Abdominals, epigastric and clonus, 0.

Eyes: Normal in every way except spontaneous nystagmus of the first degree on turning eyes right or left.

DISCLOSURES POINT TO A POSSIBLE PRESENCE OF PITUITARY DISEASE.

Further study of this case was interrupted by a sudden onset of pain in the left ear, followed by a spontaneous discharge. It was then that the patient was referred to me.

She came into my office with a staggering gait, nauseous and vomiting, a temperature of 101.6° F., and complaining of pain and discharge from the left ear.

Past History: Colds in the nose every year, lasting for about three weeks. She is not aware of ever having had any trouble with the ears. Nausea, vomiting and dizziness for the past four weeks Complains of no headache.

Physical examination revealed a moderate serosanguineous discharge, under a slight pulsation from the left ear; some sagging of the superior-posterior canal wall; marked tenderness over the left mastoid tip and antrum.

Nose: Moderately deflected septum, with a turgescent nasal mucous membrane and slight watery discharge.

Throat: Anterior and posterior pillars injected; chronically diseased tonsils of moderate size.

For the following two days the temperature ranged from 100° to 102° F. Patient was somewhat restless, vomiting which was at times projectile in character.

Eyes: Rotary nystagmus with the quick component towards the diseased side and slight vertical nystagmus on looking upwards was present.

Ears: The discharge from the left ear was more profuse and under marked pulsation. The mastoid tenderness increased, and she complained of slight frontal headache.

X-ray of the mastoid and nasal accessory sinuses were taken with some difficulty as the patient was unable to hold her head steady because of nausea and vomiting. The radiographic picture was that of a pneumatic mastoid with cloudiness and some destruction of cells on the left side. The right maxillary antrum showed opacity.

With the noise apparatus in the right ear, whispering was heard 4 inches from the left ear; lateralized to the left ear. The otoscopic picture and hearing tests were normal in the right ear.

The patient was admitted to the Brownsville and East New York Hospital on Dec. 23, 1930.

Blood count showed R. B. C., 3,200,000, with hemoglobin, 76 per cent; W. B. C., 12,800, with 82 per cent polys.

A diagnosis of a coalescent mastoiditis, possibly of long duration, was made and the intracranial condition was considered either as a complication of mastoiditis, or concomitant with a neoplasm somewhere in the cranial cavity. Encephalitis (ambulatory) was also considered.

A simple mastoidectomy was advised, with exploration of the dura, if indicated at the time of the operation.

The patient was operated upon the same day, with the following findings: Pneumatic mastoid of fairly large size, hard cortex, bleeding bone throughout, a few drops of pus in the antrum, granulations and necrosis of the cells at the tegmen antri, at the tip, near the facial ridge, and over the sinus plate posteriorly at the region of the emissary vein. There was no exposure of the dura or sinus. However, an exposure was purposely made, revealing a normal, blue-looking sinus. In view of the meager pathology, more extensive explorations were then thought inadvisable. Cultured pus from the ear yielded Gram negative bacilli (probably pyocyaneus). Cultured pus from the mastoid yielded Gram positive cocci.

There was practically no post-operative reaction, the temperature ranging from 99° to 100° F. for the next three days. Patient comfortable; no spontaneous nystagmus; no nausea and no vomiting for the first time in the past five weeks. However, on the fourth day postoperative conditions turned to the worse. Her temperature rose to 102° and on the sixth day reached 104° F. The patient complained of pain in the left side of the face, heaviness and burning in the

eyes and slight frontal headache. She was very restless at night, vomited about three or four times a day, once with bile and small blood clots, and was occasionally very drowsy.

Eye examination by Dr. John Bailey showed a progressive blurring of the right disc, with a doubtful haze in the left. The pupils reacted to light and accommodation. There were no ocular palsies, but a pseudonystagmus on turning the eyes to either side.

There was no appreciable change in the daily blood count. A blood culture after 72 hours showed no growth.

Physical examination on Dec. 27, 1930, by Dr. Samuel J. Kopetsky reads as follows: "When lying flat, spontaneous nystagmus when eyes are turned to the right, also rotary nystagmus when eyes are fixed upwards; when upright has vertical nystagmus. Changing position causes nausea and vomiting. The left palpebral fissure is smaller than the right. There is a slight deviation of the uvula to the right. There is a slight flatness of the left side of the face. She hears with the left ear when the noise apparatus is in the right. Lateralizes to the left. Knee jerks are active. There is no Brudginski, no Kernig. The abdominals (superficial) are absent. *Impressions:* From the history of a transient facial, nausea and vomiting and physical examination, I feel that we are dealing with: *1*. Perilabyrinthitis; *2*. petrositis (prodroma of meningitis); *3*. cerebellar abscess, in order of probability."

On Dec. 28 a neurologic examination showed a stronger Babinski on the right, sluggish knee jerks with a bilateral, exhaustible ankle clonus, pastpointing to the left, nystagmus with quick component to the left. There was a pseudonystagmus when the eyes were turned to the extreme right, also a rapid vertical nystagmus. There was no nuchal rigidity, but dizziness and nausea when turning from side to side.

Spinal puncture yielded 8 c.c. of clear spinal fluid under normal pressure, and showed 5 cells per c.m.m., with negative serological and bacteriological findings.

The mastoid wound was kept wide open. There was a moderate amount of discharge, with healthy granulations, and scanty discharge from the ear. On dressing the wound, moderate pressure was exerted over the horizontal semicircular canal, but no nystagmus or dizziness was elicited (fistula test negative). For the next two days there was a marked improvement. Except for a slight cough and frothy sputum, the patient was generally comfortable. The temperature ranged from 100° to 101°; respirations, 20 to 22; chest exami-

nations negative, but there was a very profuse, fetid discharge from the wound. The following three days, however, the patient presented a different picture. The temperature rose gradually to 104.4° F., preceded by chilly sensations. While mentally alert, the patient was drowsy at times, face flushed, dizziness, nausea and vomiting. The eyes were dull and heavy. She complained of slight left temporofrontal headache, severe pain in the left eye and on the left side of the occipital region. The nystagmus to the left increased and there was a vertical nystagmus. The patient resisted flexion of the head, but there was no actual nuchal rigidity. There was a tender and swollen spot about an inch in diameter over the left side of the occiput. There was also marked tenderness on pressing of the left eyeball. The mastoid wound and ear were profusely discharging. Exploration of the wound by probing for necrosis or exposures of dura resulted in negative findings.

The day following, however, there was a general improvement and thus, the picture was constantly changing.

On Jan. 2, 1931, Dr. Byron Stooky, after going over the case thoroughly, listed his observations as follows: "Nystagmus in both lateral and upward gaze, with slow component to the left. There were no extraocular palsies. The left fundus showed some blurring of the disc and the right showed some atrophy. There was a slight left facial weakness of the lower type. There was an adiadokhokinesis of the left upper extremity. Finger to nose test was well performed with the right, and with some awkwardness and swaying on the left side. Heel to knee and toe tests were done fairly on both sides. There was a bilateral clonus, more marked on the left than on the right. A bilateral Babinski was found. The abdominals could not be elicited (a fat abdomen in a young girl who had not borne children). The sensory examination was negative. There was no cortical anesthesia."

He further concluded that: "The history, combined with the neurological examination, seems to present a left cerebellar lesion of six to seven months duration or more. This is difficult to explain on the basis of an infection without the history of a source of invasion or of a pre-existing ear condition. I, therefore, am inclined to believe that the present acute infection is an intercurrent condition engrafted upon an already existing cerebellar neoplasm. However, a cerebellar abscess cannot definitely be excluded.

"My suggestion would be that the otologist do everything to obtain adequate drainage from the ear and that the patient be watched neurologically for evidence of a brain abscess, at which time a sep-

arate trephine hole be made over the cerebellum to determine the presence or absence of an abscess."

For the following three days, the condition of the patient was about the same. The temperature ranged from 100° to 104° F., the pulse, 92 to 94; respiration, 20 to 24. The nystagmus was somewhat jerky, not vestibular in character. She complained of severe pain in both eyes, slight frontal headache, pain and tenderness below the occiput on the left side. The mastoid wound and ear were discharging moderately, no nausea and no vomiting.

From Jan. 6 to Jan. 10 there was a gradual and noticeable improvement. The patient was cheerful and comfortable. There was only a pseudonystagmus to the left. The wound was granulating and the ear slightly discharging. No pain or headache, but a slight tenderness of the occiput. The hearing improved considerably. With the noise apparatus in the right ear patient heard whispering at concham over the bandaged left ear. There was still some awkwardness on attempting to touch the tip of her nose with her left hand. When sitting up with eyes closed there was no swaying or feeling of falling to either side.

Eye examination by Dr. John Bailey on Jan. 6 showed no gross defect in the visual field. The right disc was still blurred, both as to detail and margin. The left disc was hyperenic and a slight haze was still present. On Jan. 10 there was again a turn for the worse. The patient was drowsy and listless, complaining of pain in the left arm, and very severe pain in the left eyeball. The latter was somewhat protruding and apparently swollen. The left cheek was flushed. She vomited large amounts of undigested foods, occasionally greenish fluid. Marked spontaneous nystagmus reappeared. The temperature was 102° and on Jan. 11 reached 105°. The mastoid dressing was again saturated with pus, and on close examination the pus was seen to come from the ear, the antrum and the region of the semicircular canal. Probing was abstained from, although it was very tempting. At about 5 p. m. the same day the patient perspired quite freely. The temperature dropped to 101° and gradually came down to normal, where it remained until the patient was discharged from the hospital on Jan. 23, for follow-up treatment. Another favorable sign was the appearance of herpes on the left side of the lower and upper lips.

A note by Dr. Irving Sands, the neurologist, of Jan. 12 reads as follows: "Today there is a faint nystagmus on looking to the left with rapid component to the left. Pupils react promptly. Eye grounds show slight vascularity of nasal margin. All abdominals are

absent. There is a bilateral Babinski. There are cerebellar signs in the left upper extremity. The picture is constantly changing. I am inclined to favor a left cerebellar lesion—cystic in nature. However, a pituitary cyst is also a strong possibility."

On Jan. 17 the patient was allowed out of bed for one hour. The ear was dry, wound slightly discharging and hearing approaching normal. The nystagmus completely disappeared; no dizziness, no headache, no swaying to either side (Romberg test negative); finger to nose test performed equally on both sides; no Babinski. However, she developed a bradycardia with a pulse rate of 46 to 56 per minute. This alarmed me somewhat, but it lasted for only two and one-half hours and then returned to 68-72 and remained thereabouts.

The wound was completely healed by March 19, when she showed no signs of abnormality whatsoever. Her second neurologic examination on Feb. 17, by Dr. Stooky, was essentially negative; the fundi were normal, no nystagmus was present. There was no longer an adiadokhokinesis of the left upper extremity. The gait and station were entirely normal. He advised, however, another examination within three to six months.

A basal radiograph by Dr. Henry K. Taylor showed the tip of the left petrous bone partly decalcified, while the outer half of the petrous portion was pneumatized. There was no evidence of destructive petrositis.

Discussion: This case is presented primarily for its diagnostic interest because the numerous neurologic manifestations do not offer a definite clinical entity. The following were considered possible, in the order of their probability: *1.* Encephalitis, complicated by mastoiditis. *2.* Petrositis. *3.* Cerebellar tumor, complicated by mastoiditis. *4.* Cerebellar abscess. *5.* Multiple sclerosis, complicated by mastoiditis. *6.* Pituitary tumor (cystic in nature), complicated by mastoiditis. *7.* Neurosyphilis, complicated by mastoiditis. *8.* Perilabyrinthitis.

1. Encephalitis: In a symposium on encephalitis Dr. Henry A. Riley[1] truly remarks that the diagnosis of encephalitis has often served as a refuge to cloak our ignorance or inability to reconcile the results of an examination. The facile ability of this disease to ape any of the well known syndromes of nervous involvement is so omnipresent that the temptation is well-nigh irresistible to call any puzzling, unexplainable combination of symptoms of neural character, encephalitis.

As a rule the diagnosis depends on the presence of a low grade infectious process, associated with lethargy, diplopia, headache and

most important of all, evidence of the dissemination of the lesion throughout the nervous system. The diagnosis of the chronic manifestations usually depends on the identification of some influenza-like infection, which may have preceded the more slowly developing symptomatology of the disease by an interval which may vary between six months and seven years.

To substantiate our impression of encephalitis—the patient was subject to frequent colds, one of which might have been the etiologic factor resulting in the bizarre symptoms. *1.* Abnormal gain in weight (hypopituitary type) (Frohlich)(?). *2.* Numbness of the lower half of the body. *3.* Tingling sensation of the left lower and upper extremities. *4.* Facial paralysis, which appeared, disappeared to reappear, then subside. *5.* Transient form of diplopia. *6.* Diminution of mental acuity. *7.* Vomiting. *8.* Ataxia. *9.* Adiadokhokinesis. *10.* Horizontal and vertical nystagmus, not labyrinthian in character. *11.* Bilateral Babinski and absence of abdominals.

However, headache, the most important symptom, practically always present in encephalitis, was here prominent by its absence. Some of the other neurologic manifestations, such as vomiting, nystagmus, pain in the arms, pain in the left eye, seemed to appear and disappear as the condition of the mastoid wound and ear varied.

2. Petrositis: Let us assume that our patient, following one of her colds about a year or so ago, had been afflicted with one of those painless ear infections, as frequently occur in the streptococcus mucosis type. The middle ear condition cleared up, while the infection slowly spread through the peritubular or sublabyrinthian cells toward the petrosal tip, then rupturing through the posterior surface of the petrous bone. While this might occur, it is unlikely, because of the greater density of the bone in this region. The infection could have passed through the Fallopian canal to follow the course of the facial nerve backwards by way of the internal auditory canal to the internal auditory meatus, and there in the posterior fossa form an epidural abscess.

The tingling sensation of the lower half of the body; the numbness of the left upper and lower extremities; the adiadokhokinesis of the left upper extremity; the transient facial and abducens; the nystagmus; the ataxia and persistent vomiting, especially in the absence of headache, is highly significant of a lesion in the posterior fossa. These may also resemble pituitary tumor pressure.

As long as the lesion was confined below the tentorium, none of the branches of the fifth nerve were involved.

Let us now assume that the epidural abscess, instead of breaking through the dura into the cerebellum and forming an abscess, as described by Druss and Friesner[2] in their case; or into the bulbar or chiasmal cisterna, causing a generalized meningitis, as related by Eagleton[3] in his case; *that in our case,* the collection of pus possibly retrogressed through the internal meatus into the petrosa, through the sublabyrinthian cells into the middle ear and antrum, *causing* an exacerbation of the otitis; *this time,* with pain and spontaneous rupture of the drum, mastoid tenderness and temperature.

At this stage the tension was relieved by operation, resulting in the amelioration of some of the symptoms. For three postoperative days the patient stopped vomiting, temperature subsided, nystagmus practically disappeared, etc. However, the infection, in its spread, also advanced mesially toward the petrous tip, causing inflammatory reactions at the region of the Gasserian ganglion, causing the deep-seated eye pain, temporofrontal headache, temperature, etc.

One of the two cases presented by Eagleton, which came to post-mortem examination, showed the petrous apex to be the seat of the caries which had eroded both the anterior and posterior surfaces of the pyramid. The necrosis on the posterior surface spread beyond the level of the bone and into the bulbar cisterna, causing a meningitis.

Lange[4] states that in most instances the pus in the petrous tip drains into the middle ear through the channel created by the perilabyrinthian cells. In two of the nine cases reported by Kopetsky and Almour[5] the pus drained out through the avenue of invasion.

If our case is one of petrositis this must have taken place. Had the diagnosis been definitely established the operation devised by Ralph Almour would have been the proper procedure.

3. Cerebellar Tumor: Mensberger[6], in 1927, described a case which showed marked intermittent symptoms over a period of 14 years. It began with dizziness, then vomiting, attack of right-sided weakness, which after a while almost cleared up entirely. Nine years later headache developed; then weakness of the seventh nerve on the right side appeared, disappeared to reappear. In the tenth year a tetraplegia set in, lasting only a few days. Following this double vision, bilateral and vertical nystagmus and weakness of the right side of the face again appeared. There were diminished reflexes, double Babinski and bilateral ataxia. The right ear showed diminished hearing. Sudden death followed a convulsion. Autopsy revealed a right cerebellopontile angle tumor, which microscopically was diagnosed as fibrosarcoma. While cerebellar tumor is a far-

fetched possibility, we had in our case, nevertheless, to bear such a possibility in mind. The mastoid condition would then have to be considered as an intercurrent condition.

4. Multiple Sclerosis: Winkelman and Eckel[7] in reporting four cases lay stress on the similiarity of many of the clinical features of multiple sclerosis and cerebellopontile angle tumor, especially in borderline cases. They emphasize the importance of the Barany test as a help to differentiate between the two.

5. Cerebellar Abscess: There is a possibility that such an abscess could rupture spontaneously through the middle ear, possibly through the same route as in the petrositis theory.

6. Pituitary Tumor: A pituitary tumor, especially cystic in nature, although unlikely, is not to be ruled out.

7. Neurosyphilitis: This was ruled out by negative blood and spinal Wassermann and negative history.

8. Perilabyrinthitis: If this patient had a petrositis then there was a perilabyrinthitis, but not involving the peri-or endolymphatic spaces, as shown by functional tests and the absence of forced position.

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RESIDUAL HEARING AFTER RADICAL MASTOIDECTOMY.*†

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The data herein presented represents observations upon an unselected group of patients with radical mastoidectomies, who presented themselves in the private practice of Dr. Matthew S. Ersner during the interval between June, 1930, and April, 1931. No case has been omitted. Some of these had been operated upon years before by surgeons unknown to us, others recently by one of us.

It is our belief that theoretically in any given case of chronic suppurative otitis media, the radical mastoid operation should, if given proper postoperative care, result in either improved hearing or at least cause no further hearing loss than was present prior to operation. If this be true, then one contraindication for the operation, *i. e.*, the presence of a reasonably high level of hearing, should no longer be further considered. The middle ear involved in a long-standing suppurative process usually presents a thickened, partially destroyed, functionless tympanum. The tympanic cavity itself contains a conglomeration of partially necrotic granulation tissue, debris, desquamated epithelium, necrotic bone and polypoid tissue bathed in pus. The ossicles are at least partially destroyed, imbedded in masses of granulation tissue, bound down by adhesions, with loss of continuity of the ossicular chain and ankylosis of the ossicular articulations. The footplate of the stapes in the oval window is often fixed by fibrous tissue, and the round window covered with products of the inflammatory reaction. The disease itself is a focus of infection, in some instances causing loss of auditory nerve function, because of the toxic products, resulting not only from bacterial growth but also from the decomposition of protein matter within the tympanic recess.

A carefully performed radical mastoid operation removes the functionless drum head, inflammatory tissue in the middle ear and necrotic ossicles, frees the stapes from most of the factors responsible

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for its functional inefficiency, renders the round and oval windows capable of expanding and retracting, and removes the toxic element universally present in chronic suppurative otitis media. It is true that anatomical results of the operation vary considerably, but by far the great majority of these cases present postoperatively a middle ear better able anatomically to permit the passage of sound waves to the stapes and oval window. Theoretically at least then, hearing should be improved. When a middle ear has suppurated for many years we cannot hope that the lever effect of the drum membrane and ossicles will be present to intensify sound. It is not reasonable to expect that the physiological function of the middle ear and its contained structures will continue to be active. On the contrary, it becomes a structure actually acting as a detriment to the passage of sound. The amount of residual hearing in chronic suppurative otitis media is dependent upon how little or how great a hindrance the middle ear has become. The question ceases to become one of relative functional efficiency, for none is present.

The removal of this detriment to hearing (the inflammatory products and degenerated ossicles of the middle ear) lays open a channel through which sound waves can pass unimpeded except for the presence of a layer of epithelium, or possibly a thin layer of fibrous tissue covered by epithelium. It is true that the advantages of the functional efficieucy of the drum membrane and ossicles are no longer present, but in the great majority of cases there is every reason to believe that functionally, at least, they are not of any great value prior to operation. We are thoroughly convinced that even though certain chronic suppurative middle ears present a minimum loss of hearing, this residuum is not dependent upon functional efficiency of the middle ear structures, which function we believe to be destroyed early in the course of the disease. The relatively high level of hearing in these cases simply indicates that the middle ear is less of a detrimental factor than in the remaining instances. This deduction that the functional efficiency of the middle ear structures is entirely lost early in the course of chronic suppurative middle ear disease results from our many times repeated observation that almost without exception these cases are Rinné negative, with normal or only slightly prolonged bone conduction. In other words, the functional efficiency of the bone conducting mechanism is greater than air conduction, which in itself is evidence of loss of function of the middle ear structures. The theoretical considerations described find support in the observations of Hammond,[1, 2] to whose writings we shall again refer.

It is of considerable importance to know what effect an anticipated radical mastoidectomy will have upon hearing. The patient is anxious to know whether his hearing will be improved or suffer further retrogression. It has been our general rule to tell the patient that in all probability hearing will not be made worse and, under certain conditions, we believe it reasonable to expect some improvement. We feel justified in assuring the patient that in only rare instances does the hearing become less acute. Certain observations are valuable in determining the prognosis for hearing if a radical mastoid operation is to be performed.

First amongst these is the functional state of the cochlea and auditory nerve. In the face of definite and marked nerve involvement it is unreasonable to expect that any marked improvement following operation will take place. Various tests are of value in determining the functional state of the auditory nerve. Generally speaking, the bone conduction curve is the most valuable single method available for determining nerve efficiency. This should not be tested by a single note, but rather by a number of pitches throughout the audible scale. For this purpose we believe that the Western Electric audiometer, with bone conducting apparatus No. D80904, is, despite its shortcomings, the most practical method for making such an examination. If the bone conduction curve is normal, or nearly so, we believe that no marked degree of nerve deafness is present, although Fowler[3] points out that this is not always strictly true. This would tend to make the prognosis for hearing a favorable one. Keen[4] points out, and also quotes previous observations by Rauch, that in those cases in which bone conduction is diminished there tends to be a further diminution in hearing postoperatively. The Rinné and Schwabach, as determined by tuning forks, are similarly important, and likewise observations for deficiencies of hearing chiefly limited to either the high or low tonal range. These methods are well known and need only to be mentioned.

Second in importance in determining prognosis is the state of mobility of the stapes. A fixed stapes indicates a poor prognosis, and a movable stapes a better one. The presence or absence of stapedial fixation is difficult to determine, but there are available several methods, such as the well known Gellé test, the Lake test[5] and the Runge procedure, as described by Downey[6]. None of these are absolute but may strongly suggest the presence or absence of movement of the stapes.

A factor of prime importance is the postoperative care which can be given the patient. In those instances where patients come from

long distances and soon after operation return to communities where no specialized care is available, a good postoperative hearing result is not to be anticipated. This is likewise true of individuals unwilling to present themselves regularly for postoperative care. The inevitable result of such neglect is exuberant granulations, continued discharge and consequent hearing loss. Keen[4] stresses "ear exercise" and indicates that patients sufficiently conscientious to "practice hearing" will frequently have a better hearing result.

We agree with Keen[4] that the duration of the disease does not have any bearing on the hearing result. An ear diseased for a long time but in which the nerve is not markedly involved, with the stapes movable, the middle ear painstakingly freed of debris at operation and with suitable postoperative care will have a better hearing result than one of short duration, in which the nerve is involved, the stapes fixed, the operation poorly performed or proper postoperative care lacking. Indirectly, of course, duration has an effect, insomuch as in cases of long-standing the nerve is more apt to be involved and the stapes more firmly fixed by adhesions.

The literature upon the subject of hearing after the radical mastoid is rather scant. No single series of cases is entirely conclusive, and contradictions are numerous. However, by taking the general average of several groups presented by various authors, the result proves to be surprising, especially in view of the usual concept that this operative procedure is detrimental, rather than beneficial to hearing. Below are tabulated, in a rough way, statistics gathered from several sources:

	No. of Cases	*Improved Hearing*	*Hearing Worse*	*No Change*
L. E. White[7]	100	37	49	14
Keen[4]	60	23	19	18
Rauch (quoted by Keen)	100	34	60	
Buhe (quoted by Keen)	112	38	40	34
	372	132	168	66
Per cent		35+%	45+%	17+%

This group includes all types of cases, children and adults, those with nerve and conductive deafness, good and poor postoperative results, favorable and unfavorable cases, of almost every variety. More than half of this heterogeneous group presented hearing not further impaired as a result of the operation. More than a third of the cases presented actual improvement. Smith[8] reports one case in a child, age 10 years, in whom, following radical mastoidectomy, hearing improved from recognition of a loud-spoken voice at 4 inches to recognition of conversational voice at 12 feet. This, according to Fletcher[9], represents an improvement of 45 sensation units. Ham-

mond[2] reports several cases in which hearing for the whispered voice increased from almost complete loss to recognition of the whispered voice at 10 or 12 feet, a gain of at least 45 sensation units.

In considering that 45 per cent of this series had worse hearing after operation than before, it must be borne in mind that during the course of years between operation and final testing it is not at all unlikely that some further diminution in hearing would have occurred even under more conservative methods of treatment. We have no accurate data available upon which to draw conclusions concerning the rate of hearing loss with and without operation. It may well be that such loss as has been reported in 45 per cent of these

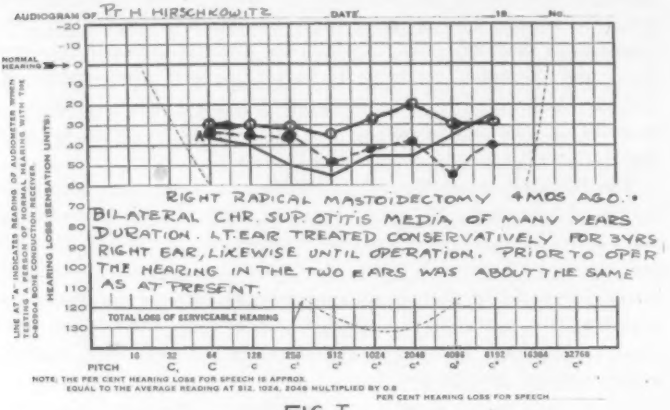


FIG. I

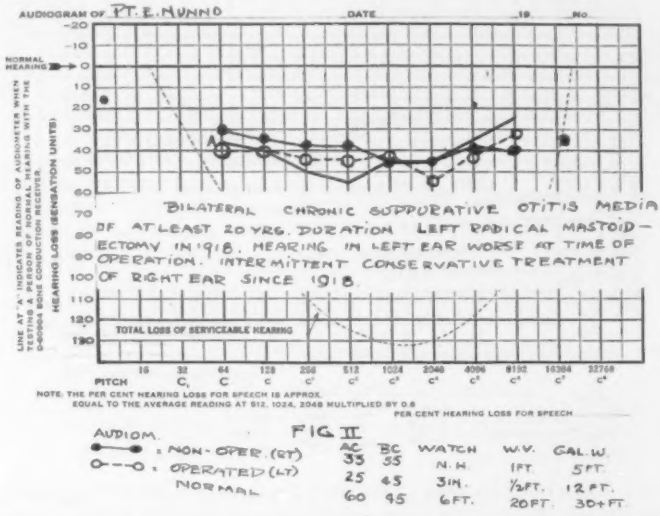
	AUDIOM.	AC	BC	WATCH	W.V.
RT. OPERATED	●—●	35	50	6 IN	2½ FT.
LT. NON-OPERATED	O—O	50	50	6 IN	3 FT.
NORMAL (GIZDEK, A. 0964)		95	55	72 IN	20 FT.

cases may have been greater if operation had not been performed. It is almost certain that in cases in which the pathological state of the middle ear is becoming progressively worse that hearing will similarly suffer, and although some further diminution in hearing may follow the radical mastoid operation, it is not at all certain that such loss may not have been greater if the operation had not been performed.

We are able to report careful detailed studies upon thirteen patients with radical mastoidectomies. The age incidence in this series is from 9 to 45. The time elapsed since surgery is from 4 months to 16 years. All the operated ears in this series are healed and dry except for one or two with slight mucoid discharge, apparently from

the Eustachian tube. Only a very few of these cases offer pre- and postoperative comparisons since most of them were operated upon before it had been customary to use standardized tuning forks, and when audiometry was not clinically available.

Certain of these patients had, when first seen, bilateral chronic otitis media of equal duration. Those cases which were operated upon in our clinic had the operations performed upon the ear in which hearing was the less acute. It may, therefore, be valuable to compare the hearing in the two ears, bearing in mind that in nearly all cases, the operated ear at the time of operation presented a greater hearing loss than the other. By this comparison some rough estimate may be obtained relative to the effect of surgery upon hearing.



Case 1: H. H., age 24 years; bilateral chronic suppurative otitis media since infancy. Left ossiculectomy, Feb. 3, 1925. Right radical mastoidectomy, Oct. 13, 1930. Both ears now dry. Hearing tests, Feb. 25, 1931. Results indicated in Fig. 1.

Case 2: E. N.; Bilateral chronic suppurative otitis media for many years (duration unknown). Left radical mastoidectomy performed in 1918. Results indicated in Fig. 2.

Case 3: S. W., age 42 years; bilateral chronic suppurative otitis media of 32 years duration. Left radical mastoidectomy in 1914. Results indicated in Fig. 3.

Case 4: B. B., age 35 years; bilateral chronic suppurative otitis media of 34 years duration, beginning after diphtheria in infancy. Left radical mastoidectomy in 1929. Results indicated in Fig. 4.

Analysis of these four cases indicates that in one case (Case 1) the hearing of the operated ear is definitely worse than the non-operated ear. In this case operation was performed four months prior to testing and the hearing was preoperatively worse in the ear to be operated upon. There has been no diminution in hearing in the operated ear in comparison with the preoperative level.

The other three cases have about equal hearing in the operated and nonoperated ears. Prior to operation the hearing in the operated ear was as poor as or worse than the hearing in the nonoperated

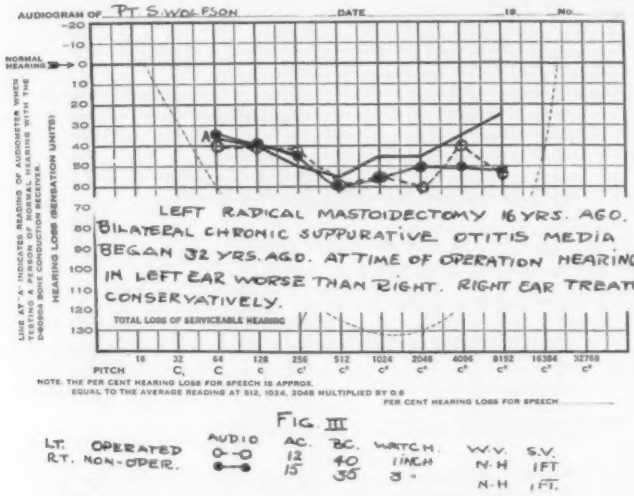


FIG. III

ear. Therefore, in these cases, each presenting bilateral suppurative middle ear disease, the suppuration in each instance being of equal duration on the two sides, radical mastoidectomy has not resulted in greater loss of hearing than more conservative therapeutic measures.

The value of these observations, rather than pre- and postoperative comparisons on the same ear, is that in such a series as this, the hearing loss which would have occurred since the time of operation, had no surgery been performed, is in some measure taken into consideration by comparison with the opposite (or nonoperated) suppurating ear.

The second group for comparison presents hearing in the same ear pre- and postoperatively. There are again four patients in this

group. In some instances the data is incomplete, but all available observations are presented.

Case 5: H. H., age 24 years; right suppurative otitis media since infancy. Radical mastoidectomy, Oct. 13, 1930. Postoperative hearing approximately four months later. The ear is dry and healed and epithelialization is complete. Results indicated in Fig. 5.

Case 6: S. C., age 16 years; left suppurative otitis media since infancy, following measles. Left radical mastoidectomy, Nov. 1, 1930. Hearing tests about three months later, before healing had been complete. The patient did not present herself for postoperative

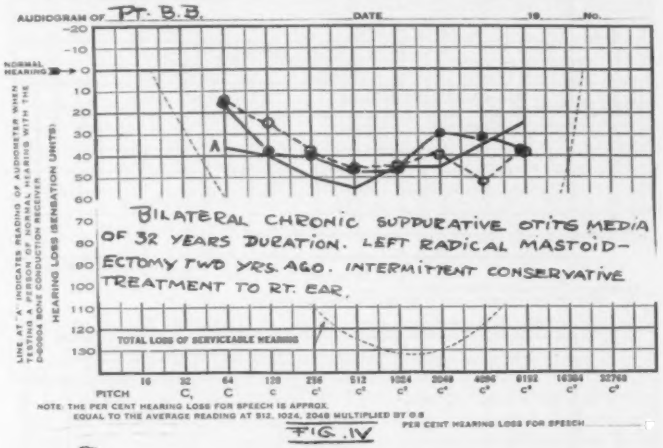


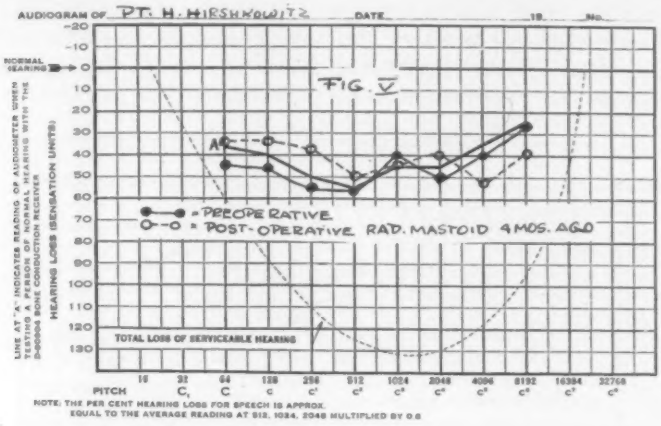
FIG. IV

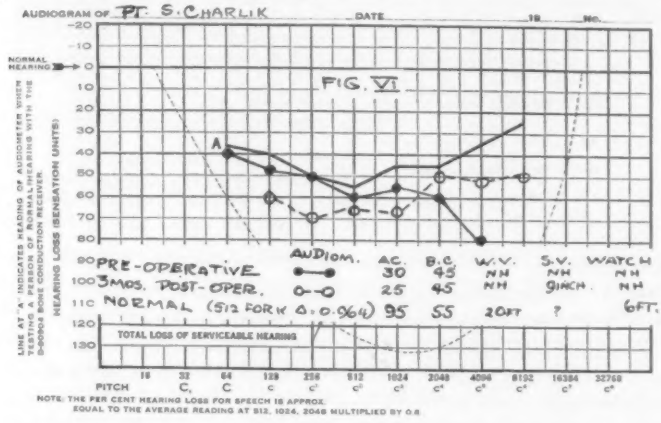
	AUDIOM.	AC	BC	G.W.	WATCH	W.V.
RT. (NON-OPERATED)	●—●	30	50	5FT.	9IN.	11 FT.
LT. (OPERATED)	o—o	40	45	24FT.	9IN.	6 FT.
NORMAL. 512 FORK		95	55	30+FT	72 IN.	20 FT.

care from Dec. 6, 1930, to Jan. 25, 1931, and at the time of postoperative testing the cavity presented a marked overgrowth of granulation tissue and moderate discharge. Results indicated in Fig. 6.

Case 7: L. F., age 8 years. In June, 1930, the patient developed an acute suppurative otitis media and mastoiditis, for which a simple mastoidectomy was performed. During the third week of convalescence, symptoms suggestive of a localized meningitis developed, for which a radical mastoidectomy with a decompression over the middle and posterior fossa was performed. Subsequent recovery was uneventful and the ear was dry and healed in about eight weeks. Hearing tests were performed Dec. 4, 1930, five months after the radical mastoidectomy. Results indicated in Fig. 7.

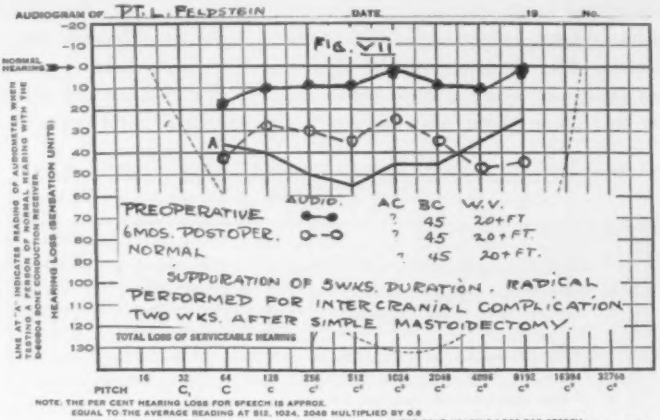
Case 8: P. G., age 27 years; left chronic suppurative otitis media of unknown duration, but it has been several years since onset. Radical mastoidectomy, Dec. 5, 1930. Postoperative hearing tests, January, 1931. The ear is well healed, but a discharge from the Eustachian tube persists. Epithelialization is complete. Results recorded in Fig. 8.

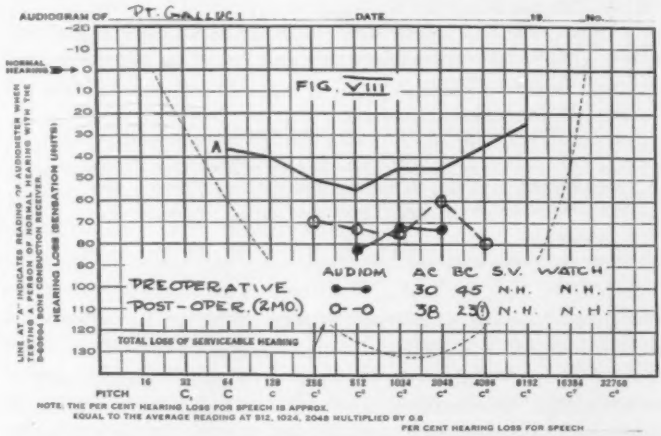




It is thus seen that in these four cases, two (Cases 6 and 7) present some diminution in hearing after operation; one (Case 8) is definitely improved, and the fourth (Case 5) shows no change in audiometer level, but some improvement according to other methods of testing.

The majority of this entire group of 12 cases present nothing of statistical value other than the observation of their present level of hearing. In order to present some basis for comparison we have tested 12 consecutive unselected cases of chronic suppurative otitis media upon whom no operations had been performed. These latter

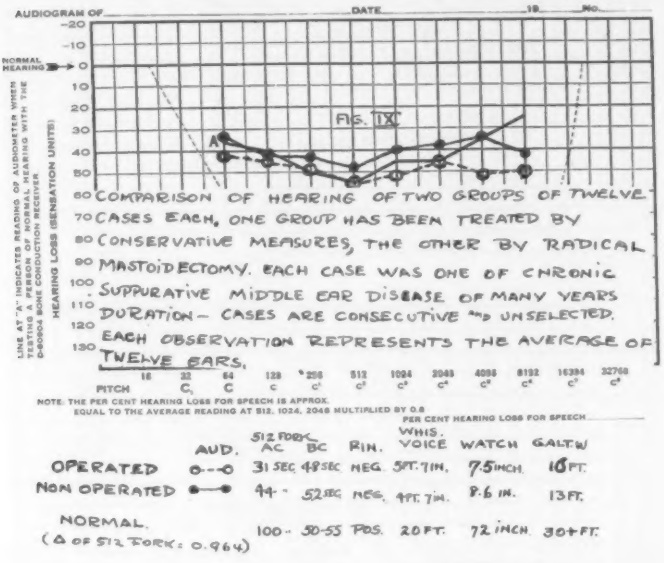




cases have been treated conservatively and it may be of some slight significance to compare these two groups to indicate in some fashion the relative hearing resulting from conservative and radical methods of treatment. Since there is no relationship other than similarity of disease and duration in these two groups, averages rather than indi-

vidual cases are sufficient for comparison. One case of the radical mastoid group is omitted because a labyrinthitis complicated the picture.

Fig. 9 indicates average hearing levels for each group of 12. The audiometer level of the nonoperated group is slightly higher than the radical mastoidectomy group and, similarly, other methods of testing, except whispered voice, show a slight advantage in favor of those treated conservatively. The operated group, however, hear the whispered voice a little better than do the nonoperated cases. On the whole, those who have not been operated upon present a



slightly greater acuity of hearing. It must, however, be remembered that cases are often selected for conservative treatment if the hearing is not too greatly impaired, whereas those selected for radical mastoidectomy usually have when first seen a greater degree of hearing loss. The comparison of our two groups demonstrates that the difference in hearing following the two methods of treatment is very small, and certainly not enough to prejudice us against the radical mastoidectomy if it seems to be indicated. The prognosis for hearing is apparently about the same regardless of the method of treatment.

The results of our observations, taken together with previous reports in the literature, therefore, indicate that in 35 per cent hearing following radical mastoidectomy is improved. In other cases a loss may follow (45 per cent), and in the remaining instances no great change in either direction follows upon this operation. In the majority of cases, therefore, radical mastoidectomy does not have a detrimental effect upon hearing. This is apparently no great variation in the level of hearing after several years, regardless of the mode of therapy.

SUMMARY.

It is pointed out that, theoretically, the radical mastoid operation should not be detrimental to hearing.

Three hundred and seventy-two cases of radical mastoidectomy, gathered from the literature, present 35 per cent improved hearing, 45 per cent with further loss of hearing, and 17 per cent with no change.

Several cases from the literature are described in which radical mastoidectomy resulted in an improvement of hearing amounting to at least 45 sensation units.

Four cases are presented, each with bilateral otitis media and both sides of similar duration. One ear of each case was treated conservatively, the other by radical mastoidectomy. The poorer hearing ear was selected for operation. The end-result so far as hearing is concerned reveals that in three cases there is no great difference in hearing between one ear and the other. The fourth case, four months convalescent, presents a greater loss in the operated ear, but this relationship existed prior to operation, and no further loss in hearing followed the radical mastoidectomy.

Four additional cases are presented and the hearing level compared before and after operation. Two of these four cases presented additional loss of hearing after operation, one is definitely improved, and the fourth shows no considerable change in either direction.

Two similar groups, each of 12 consecutive cases of chronic suppurative otitis media, are compared insofar as hearing is concerned. One group had been treated conservatively; the other by radical mastoidectomy. The nonoperated group presented slightly better hearing, but it is pointed out that cases selected for radical mastoidectomy usually suffer a greater hearing loss prior to treatment than those who are treated conservatively. The difference in hearing level is, at any rate, very slight.

CONCLUSIONS.

1. Theoretically, the hearing in chronic suppurative otitis media should not be detrimentally influenced by radical mastoidectomy.

2. Actually, the majority of cases so treated have as a result of the operation no further loss in hearing.

3. The hearing level of cases of chronic suppurative otitis media is, from the standpoint of averages, not appreciably lower when treated by radical mastoidectomy, than when treated by conservative measures.

4. A relatively high level of residual hearing is not a contra-indication to radical mastoidectomy.

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240 S. Doheny Drive, Beverly Hills.

BILATERAL LABYRINTHINE HEMORRHAGE IN A CASE OF APLASTIC ANEMIA.

Dr. Michael Rosenbluth, Bronx, N. Y.

Before reporting the case it would be interesting in a few words, to recall what is meant by aplastic anemia. Aplastic anemia is that form of anemia which results in a replacement of the bone marrow by fat, so that there is no formation of red or white cells or platelets. The condition is characterized by the absence almost completely of granulocytosis, hardly any platelets, marked reduction of red cells, and no attempt at the regeneration of cells. Finally, when regeneration begins to take place, the fact is shown by the appearance of normo- or megaloblasts, myelocytes or myeloblasts, polichromatophilic red cells, anisocytosis and poikilocytosis.

Etiology: Idiopathic (congenital), X-ray workers, benzol workers, radium workers, syphilis, toxic drugs (in this case, arsenic).

Clinically, the chief characteristics are hemorrhages of various degrees and intensity from various parts of the body, with marked anemia.

History of the Case: J. W., male, white, age 48 years, was brought in an ambulance to the Lebanon Hospital early in the morning of Sept. 2, 1929. He was at the time almost exsanguinated from marked epistaxis; he was almost stuporous from loss of blood, and on attempting to raise his head he developed syncope. His nose was packed, and bleeding at the time was controlled.

Previous History: He has had syphilis for the past 10 years, during which time he has had numerous courses of antiluetic treatments. Six weeks ago he noticed a small, indurated sore on the penis. He got 1 gm. bismuth for two weeks and ½ gm. arsphenamin once a week for about five weeks. Five weeks ago he noticed that he was becoming pale, dizzy and weak, but no definite pains. Four weeks ago bleeding developed from the gums and, on going to a dentist, one of his right upper molars was removed. Four days previous to admission to the hospital he had a severe nasal hemorrhage, which was controlled only after a long time, with much difficulty. This hemorrhage from the nose kept recurring from time to time during the four days up to admission to the hospital. At each hemorrhage

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there was a large amount of blood lost before the hemorrhage was brought under control.

On admission the physical examination showed a well built man, somewhat stuporous, probably from loss of blood, and very markedly anemic. Pulse was 110; temperature, 103°. Teeth and gums still showed signs of bleeding. Both sides of his nose were packed.

He received a transfusion by the citrate method of 500 c.c. His blood was Type 2. Blood examination showed: Hemoglobin, 26 per cent; red blood cells, 1,200,000; white blood cells, 2,000; small lymphocytes, 69; large lymphocytes, 4; eosinophiles, 2.

Most of the red blood cells are microcytes and achromatic. Very little poikilocytosis.

The history of the case, the physical examination and the blood examination point to an aplasia of the bone marrow system due to arsenic.

On Sept. 3 the packing was removed. There were numerous fresh hemorrhages in the left eyeground, and in the region of the left iliac crest. There were several small petichiae and one subcutaneous large hemorrhage.

On Sept. 7 Dr. Percy Fridenberg examined the eyes again and reported the right eye showed vessels irregular and the veins markedly dilated; left eye, extensive vascular changes of the retina, with numerous hemorrhages in the course of the vessels, mainly along the veins.

Sept. 11, there were some moderate-sized submucous hemorrhages on the hard palate.

Sept. 13, he complained of pain in his ears. Both drums were congested. The X-ray showed normal mastoids.

Sept. 14, the right ear ruptured spontaneously with bloody discharge, also there had developed a bulging of the superior posterior quadrant of the drum. In addition, there was a slight bulging, palpable mass, just below the ear, from just above the mastoid tip, down along the sternomastoid, painful on touch. The left ear showed a picture similar to that on the right, but there was no mass outside. The question arose whether we were dealing with a mastoiditis in the right ear. One might have easily supposed so, because there was profuse discharge, there was marked tenderness over the mastoid, there was the swelling of the mastoid, beginning at the tip and extending downward, and temperature.

Sept. 15, tenderness over both mastoids, the right greater than the left. Both drums were opened more widely, admitting a freer

drainage in the hope that this would help resolve the pathological condition.

Sept. 16, an ulcer developed on the uvula. Smear showed that there was a Vincent's angina.

Sept. 17, uvula appeared gangrenous.

Sept. 23, Dr. Isaac Heller reported edema and tenderness over his right mastoid, the lower one-third on his uvula had sloughed off; he advised against operation. This ultimately turned out to have been very proper advice. The swelling became ecchymotic and gradually disappeared. Evidently it had been a deep, subcutaneous hemorrhage and not the swelling of mastoiditis, which for a time it was suspected of being.

Sept. 18, on addressing the patient he answered with a blank stare. He could not hear at all. When I examined him I found a large perforation in each drum, with profuse, purulent discharge. Large granulations were protruding through each opening. He could not hear any tuning forks in either ear, by ear or bone conduction. On doing a caloric test with cold water on both ears, there was not produced any response whatever after irrigating each ear for about five minutes. No nystagmus could be elicited from irrigation of the ear, even with the eyes turned to the optimum position to elicit this response. Thus, on irrigating the right ear, which should have elicited a nystagmus to the left, there was no shadow of such a response, even with the eyes turned towards the left; and similarly no response from the left ear with the eyes turned to the right.

The absence of sound perception on bone and air conduction already suggests deafness of central origin (nerve or cochlea). In addition, absence of response to caloric test speaks for hemorrhage into the inner ears, destroying the functions of both cochlea and semicircular canals on both sides. In middle ear involvement alone, deafness is never so marked and a nystagmus would be elicited because the semicircular canals would still be functioning.

Nov. 17, Dr. M. D. Lederman advised the use of iodin powder treatment for the granulations of the middle ear. This was followed by a remarkable rapidity in the disappearance of granulations and in about two weeks of this treatment the granulations had almost entirely disappeared and both ears were dry.

Repeated caloric tests of both ears at various times always gave the same result. At no time was there any response in the form of a nystagmus elicited from either ear, even after prolonged irrigation.

Nov. 17, he had already been out of bed a number of days and in fair physical condition, and I decided to study his labyrinthine responses with the rotation test, and here again no response in the form of nystagmus, vertigo, past-pointing or falling could be elicited with the head at different times held in different positions in order to elicit any possible labyrinthine reaction.

Dec. 11, he was discharged. His general condition was very good. His hemoglobin was 72 per cent and his red blood cells were 4,000,000. A few days after his discharge from the hospital, I demonstrated the caloric and rotation tests on him before the clinical meeting of the Lebanon Hospital alumni. At this time, as at all other itmes, no reaction of any sort was elicited. During his stay in the hospital he received six transfusions by the citrated blood method, as follows: Sept. 8, 500 c.c.; Sept. 12, 400 c.c.; Sept. 16, 400 c.c.; Sept. 21, 400 c.c.; Sept 30, 400 c.c.; Nov. 11, 360 c.c.

During his stay in the hospital the blood was examined almost every day. As it was recognized that the bleedings from the nose in this case were not due to a local nose condition, but were the result of some blood dyscrasia, the patient was transferred to the medical service, where the case was studied and treated under the direction of Dr. William Weinberger.

On Sept. 2 his Wassermann was negative.

Sept. 8, there is is a trace of arsenic in the stool. This was present almost every day until Sept. 24, when no arsenic in the stool was present, and from this date on could not be found in the stool any more. His temperature on admission was 103°. It ranged between 102° and 103.5° until Sept. 12, then it began to drop by lysis until Sept. 20 it reached 99° and continued so.

COMMENTS.

These dead inner ears were not the result of arsenic poisoning because the arsenic had disappeared from the stool on Sept. 24, while this loss of hearing in both ears occurred Oct. 18, 24 days later. It is much more reasonable to suppose that the hemorrhages which were going on all over, which were simultaneously taking place in both eyegrounds and both middle ears, had also simultaneously attacked both internal ears. The absence of spontaneous nystagmus is accounted for by the fact that both labyrinths stopped functioning at the same time so that there was no predominance of stimuli from one side over the other. He is still stone-deaf and gives no vestibular responses to caloric or rotation tests.

Sept. 13, at the time when possible mastoiditis of the right ear was suspected, X-ray showed normal cellular pneumatic mastoids on both sides.

Sept. 16, X-ray report was the same as on Sept. 13.

Sept. 16: Hemoglobin, 21 per cent; R. B. C., 1,010,000; poilkulocytes, 35 per cent; myelocytes, 10 per cent; myeloblasts, 2 per cent; monocytes, 4 per cent; small lymphs., 46 per cent; large lymphs., 3 per cent; megaloblasts, 2 per cent; polichromatophilic red blood cells, 4 per cent. The picture in the smear shows a very slight attempt at marrow regeneration.

Sept. 3: Blood chemistry showed: N. P. N., 29; urea N., 14; uric acid, 1.7; sugar, 150; Co., 246.

BLOOD EXAMINATION.

	Sept. 2	Sept. 3	Sept. 5	Sept. 9	Sept. 12 Before Trans.	Sept. 12 After	Sept. 20
Hemoglobin	26	32	28	30	20	30	27
R. B. C.	1,200,000	1,530,000	1,250,000	2,120,000	980,000	1,000,000	1,300,000
W. B. C.	2,000	2,000	2,000	1,000	1,400	1,000	2,400
Neutrophiles	12	21	20				28
Small Lympha.	88	79	80				68
Large Lympha.							
Transitionals							
Eosinophiles							
Basophiles							
Unclassified							Myelob'asts 2%
Abnormal Cells							
Platelets							

Sept. 23	Oct. 4	Oct. 7	Oct. 31	Nov. 28	Dec. 3
In over	32	30	53	70	72
2,000,000	2,050,000	1,770,000	2,610,000	4,100,000	4,250,000
R. B. C. 210	2,600	2,700	6,200	7,900	8,200
Reticulocytes seen.		55	53	61	61
		53	30	26	27
		4%	2		
		3%	15	3	2
				10	

1018 East 163rd Street.

FATAL CASE OF PHARYNGEAL HEMORRHAGE.*

DR. AARON KAUFMAN, Boston.

This case is reported because of its unique and rare complication, which one may encounter occasionally in a large clinic.

Case Report: A female patient, age 2 years, admitted on Sept. 18, 1930, on the pediatric service with a history of swelling in the neck and fever. The history was obtained from the mother.

Present Illness: Mother states that three weeks ago baby had a convulsion. At this time mother called the family doctor, who discovered a swelling in the neck (left side). This disappeared after a few days with treatment, only to reappear again five days later. On the advice of the family physician the child was brought to the hospital.

Past History: Essentially negative. Normal breast-fed baby. Normal birth weight, 10 pounds. *Family History:* Negative.

Physical Examination: A fairly well developed and fairly well nourished infant, admitted with no signs of distress, pale fascies and skin. No skin eruption present. Head held toward the left side, respiration normal. *Skull:* Symmetrical, anterior fontanelle open, posterior closed. *Face:* Symmetrical, no facial palsy. *Ears:* Apparently normal. *Mouth and Lips:* Normal. *Nose:* Negative. *Throat:* Tongue coated, slight injection of throat, involving pillars and oropharynx. Slight swelling behind left pillar, this region appearing more prominent than the right. *Neck:* Essentially negative. No glandular adenopathy. *Thorax:* Heart and lungs negative. *Abdomen:* Negative.

Blood Picture: Aug. 19, 1930: R. B. C., 4,970,000; W. B. C., 30,000; polys., 84 per cent; lymphs., 16 per cent. Aug. 21: R. B. C., 2,540,000; W. B. C., 21,400; polys., 72 per cent; lymphs., 28 per cent; B. T., 2 minutes; C. T., 3 minutes; H. G., 55 per cent. Aug. 22: R. B. C., 4,250,000; W. B. C., 16,300; polys., 56 per cent; lymphs., 44 per cent; H. G., 60 per cent. Aug. 24: R B. C., 3,950,000; W. B. C., 21,000; polys., 25 per cent; lymphs., 71 per cent; endothelial, 4 per cent. Aug. 26: R. B. C., 3,930,000; W. B. C., 16,000; H. G., 60 per cent. Aug. 29: R. B. C., 3,010,000; W. B. C., 54,900; polys., 78 per cent; lymphs, 19 per cent; B. T., 2 minutes; C. T., 7 minutes.

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*From records of Boston City Hospital.

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Admission Note: A fairly well developed and fairly well nourished, white, anemic infant, referred to the hospital on account of fever and swelling in the neck.

Aug. 21: Two days following admission child began to bleed profusely from left ear. The aural service inserted a pack in the ear canal to stop the hemorrhage. During this procedure the eardrum was not visible on account of the profuse bleeding, which poured out in a stream and apparently came from the floor of the drum and middle air cavity.

Aug. 23: Temperature today normal. Throat injected, swelling behind left pillar appears to involve supratonsillar fossa, no pulsations felt. Another hemorrhage from the left ear arrested by packing the ear canal; 200 c.c. of blood transfused (citrated). The white cells decreased, red cells remaining about the same.

Aug. 26: Temperature normal, pulse still rapid, no bleeding visible from the ear canal except for mucopurulent discharge from the anterior-inferior region. After wiping ear canal, drum appeared gray and dull with the disappearance of normal landmarks. No visible perforation present. Bulging behind left pillar (posterior) more prominent.

Aug. 28: Throat injection has disappeared, bulging not so marked. Condition good.

Aug. 29: Patient bled profusely this evening from left ear. Transfusion performed. Early this morning the patient began to bleed from his nose, nasal pack inserted. Patient's condition poor, skin and mucous membrane pale in appearance. Aural service called and during the course of examination the mass in the throat ruptured and considerable quantity of blood and clot poured forth. Patient died while attempts at packing were being made.

During stay, patient's temperature varied from 101-104° rectally, reaching the highest on third day of entrance. Pulse varied from 136-150, and respiration from 22-36.

Autopsy Findings: Negative except for picture that goes with secondary anemia and local findings in the throat, which were as follows: Grossly specimen revealed rounded mass of soft red tissue lying adjacent to the first part of the internal carotid artery on the left side. It measures 3.5x2x1.5 c.m., causing a distinct bulging of the retropharyngeal wall. Section discloses what appears to be old blood clot lying in a small cystic area with a wall of granulation tissue, averaging 2-4 c.m. in thickness. Small blood vessels are found entering this cavity as though they had been eroded through.

Anatomical Diagnosis: Old retropharyngeal abscess with erosion

into blood vessels and secondary hemorrhage. Inspissated blood in the bronchi. Dark brown blood in the gastrointestinal tract.

It is interesting to note that the pathological anatomical examination revealed the fact that the erosion appeared to involve the first branch of the carotid which sends a branch to the tympanic cavity. Whether or not this was responsible for the bleeding from the ear is a matter of conjecture.

In looking over the literature on this subject, it is interesting to note how few and scattered the cases are. One case reported by Wylie and Wingrave, of London, in which evacuation of pus from a retropharyngeal abscess in a 24-year-old female, for dyspnea, resulted in death due to sloughing of the internal carotid. Another case reported by Franklin, of New York, of a retropharyngeal abscess in 7-year-old child, ulcerating into the left internal carotid. This was followed by a right hemiplegia with aphasia, patient subsequently recovered. In this case the mass in the postpharyngeal wall ruptured spontaneously and was controlled by a pack placed behind the posterior pillar through the nasopharynx. As the author himself states "the hemorrhage should cease spontaneously seems so highly improbable, although the nervous phenomena which followed point directly toward the establishment of the diagnosis made." The resulting complication in this case, as explained by the author was probably caused by an embolus of the terminal branch of the internal carotid (middle cerebral artery).

A more recent case, reported by F. Klug, of a retropharyngeal abscess of auricular origin in a child, age 12 years, with erosion of the carotid and death from hemorrhage 24 hours after opening the mass. Klug, F.: Retropharyngeal abscess of the auricular origin: erosion of the carotid. Ann. d. mal. de L'oreille (etc.), Par., 1904. Central London Throat and Ear Hospital: Case of retropharyngeal abscess with death from sloughing of the internal carotid; Dr. Andrew Wylie and Dr. V. H. Wingrave. Retropharyngeal abscess ulcerating into the left internal carotid with recovery; M. M. Franklin, New York.

THE DIAGNOSIS AND CONSERVATIVE TREATMENT OF SPHENOID SUPPURATION.*†

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Excellent but few contributions have been made to the literature on this subject and comparatively little original research work has been done during the past several years. The sphenoid sinus, as is well known, is obscurely located, and although it is only a small part of the respiratory mechanism it nevertheless merits special attention[1]. Suppuration may involve the sphenoid separately but is usually a part of an ethmoid involvement. Infection involving this sinus with or without posterior ethmoid involvement is most difficult and one of the rarely diagnosed of sinus infections[2]. The reason for this is apparent when one considers the mild and ofttimes vague symptom complexes, together with the almost inaccessible location of these cavities. No doubt some sphenoids with a low grade infection have escaped detection for years, having been considered merely a chronic catarrhal condition until, the sudden appearance of a severe ocular disturbance or another complication invited immediate attention.

Of all the sinus suppurations, the sphenoid is probably most dangerous because of it being surrounded by the meninges, the cavernous sinus and its contents, the carotid artery, the hypophysis, and the optic nerve and chiasma. Therefore, to avoid any serious complications from extension of infections and to expect rapid recovery, early diagnosis and proper treatment are imperative.

A. Diagnosis.

It is only by intelligent interpretation of subjective symptoms and objective physical findings that diagnosis reaches a firm foundation, and especially is this exemplified in the diagnosis of sphenoiditis. Sphenoid suppuration, with which this subject deals, is less difficult to diagnose than the hyperplastic type, although "there is one clue," as Skillern[3] has said, and that is the "appearance of the middle turbinate." The body of this structure appears slightly hypertrophied and frequently has a mottled appearance.

Today one has learned there is more to the diagnosis of sphenoid suppuration than the mere appearance of the middle turbinate. The

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diagnosis is dependent upon a scrupulously detailed history and a carefully scrutinized study of the objective physical findings, the radiographs and the cytologic washings.

I. History: The various symptoms associated with sphenoid suppuration are numerous and vary from simple headaches to the peculiar symptom complexes, some of which are seemingly unattached to the sphenoid. No doubt the morphological variations are a factor in the symptomatology of this disease[4]. So evident is this that occasionally sphenoidal suppuration may simulate intracranial lesions and because of the seriousness and magnitude of such an operation as an exploratory craniotomy, and the dangers of a sphenoidectomy, every procedure available should be used in the differential diagnosis[5]. Sphenoidal infection may occur in children, as Myerson has reported a death in a child 5 years old from meningitis in which the sphenoid sinus showed pus[6]. Shaeffer, Shea[7], and others[8] have called our attention to the development of the sphenoid in children as young as 3 years of age, consequently a sphenoidal suppuration at that age is not impossible and, therefore, one should bear in mind the possibility of a sphenoid involvement in childhood. Three such cases have come under my observation recently, corroborated by radiographs and cytologic lavage. MacGibbon, in a paper on sphenoethmoiditis, states that this condition is common in sections of Australia, stating that 50 per cent of the children of Christ Church have infection of the ethmoid and sphenoid sinuses[9]. Although I do not doubt this statement, I do believe that we have less involvement of the sphenoid in this country. Children with impaired vision as existed in my cases, in whom there is no pathology within the eye to account for the diminished visual acuity, should require an investigation of the sinuses, especially the sphenoid. Often we are surprised at the size and degrees of development of this sinus in children. Radiography in the Hirtz position and cadaveric study have demonstrated this repeatedly.

Sphenoid suppuration may be acute or chronic. Acute suppuration manifests itself clinically with symptoms suggestive of a cold very closely resembling other acute sinus infections. It may be the initial involvement or it may be an exacerbation of a chronic one. The symptoms are very similar to those of the chronic except that the attack is sudden, the headache, neuralgic pains and tightness in head are most intense and the vertigo may be well pronounced. The optic neuritis may result in a transitory blindness and the field of vision and color acuity may be so affected as to produce a temporary color blindness and tubular vision.

The history is: *1.* One of upper respiratory affection with a tendency to recurrent head colds; headaches; pain between the eyes; throat, nasal and mental symptoms; ear and ocular manifestations, and many general symptoms apparently aberrant to the sphenoid.

2. Headaches are produced as a result of toxemia, and pressure within the sinus or rarefaction from vacuum, or irritation of the Vidian nerve or sphenopalatine ganglion resulting in the "front-half" type of neuralgic headache in which the pains are typically sensory along the distribution of the fifth cranial nerve and the "back-half" type myalgic with myalgic nodes vasomotor in origin along the distribution of the occipital nerves accounting for the Vidian and Sluder syndromes[10]. *A.* Pain originating in the eye may be found radiating to the temple through to the mastoid region and behind the head to the nape of the neck[11]. *B.* The mastoid pain may become such as to simulate the pain of mastoiditis. Firrot, in 1927 cited three cases of simulated mastoiditis in which the patients were relieved by sphenoid drainage[12]. *C.* Occipital pain, so frequently a manifestation of sphenoid infection, is the result of Vidian nerve irritation in which the reflex is carried through the great deep petrosal nerve to the carotid plexus, thence to the superior cervical sympathetic ganglion to the cervical plexus and finally through the three upper cervical spinal (occipital) nerves. Occipital pain localized over the mastoid is often associated with vertigo and tinnitus, and when occurring together is spoken of by Oaks and Merrill as "the sphenoidal sinus syndrome"[13]. Bertermes, in 1925, reported a case with occipital and mastoid pain in conjunction with wry neck, in which rapid disappearance of the pain and torticollis occurred after sphenoid treatment[14]. The sphenoid discomfort varies from a heavy, steady aching to lancenating, stabbing pain, or a burning sensation; and the area described is often sensitive to superficial pressure.

3. Throat and nasal symptoms are: Coryza as before mentioned, post-nasal discharge occasionally with scab formation and constant hawking, often associated with hoarseness or aphonia; anosmia; subjective odor; parosmia; dry productive cough; clearing of throat and gagging.

4. Mental symptoms simulate those of neurasthenia, and may be severe enough to produce delirium[15]. They may be best expressed as Citelli's syndrome, which was again called to our attention by Caleciti in 1925, characterized by defective memory, somnolence, insomnia, intellectual deficiency and unwillingness to concentrate[16].

5. Ear symptoms are: Diminished hearing, tinnitus, vertigo, fullness, otalgia and splashing in ears. Sluder[17], in 1926, called our

attention to diminished hearing with fullness associated with it, and attributed it to Eustachian salpingitis resulting in catarrhal deafness. During the same year the superior cervical sympathetic ganglion syndrome described by Palmer[18] gave to the profession an explanation to account for the tinnitus and vertigo which so frequently accompanied sphenoid infection. Through irritation of the superior cervical sympathetic ganglion which supplies the nervi vasorum of the internal auricular branch of the occipital and the auditory arteries to the labyrinth, hyperemia of the organ is produced which brings about the tinnitus and vertigo. Therefore, vertigo and tinnitus are not due entirely to toxemia. Vertigo may be expressed as "giddiness" or "light-headedness," or a "feeling of falling." It is usually experienced upon motion of the head or when walking, stooping or turning.

6. Ocular manifestations are: Redness of eyes; pain in and about the eyes; asthenopia; muscle imbalance; photophobia; impaired vision from optic neuritis or atrophy; and always suggestive are the refractive states that vary with repeated examinations and little improvement. Diminished visual and color acuity and concentric contraction of the color fields often associated with central scotoma (relative or absolute), enlargement of blind spot and pallor of disc.[19, 20, 21, 22, 23, 24, 25, 26, 27, 28, 29, 30, 31, 32, 33.] One should be cautious in considering sphenoid infection as a cause of optic neuritis as thirty-four other causes have been enumerated by Vail[34]: *1.* Multiple sclerosis; *2.* acute myelitis; *3.* Leber's hereditary optic atrophy; *4.* tobacco alcohol amblyopia; *5.* diabetic amblyopia; *6.* neuritis from a gravid uterus; *7.* lactation; *8.* sinus infections; *9.* infections and toxemia; *10.* cerebrospinal; *11.* tuberculosis; *12.* typhus; *13.* ery- *13.* erysipelas; *14.* beri-beri; *15.* influenza; *16.* mumps; *17.* pneumonia; *18.* angina; *19.* meningitis; *20.* encephalitis; *21.* smallpox; *22.* measles; *23.* pertusis; *24.* vaccination; *25.* Bright's disease; *26.* severe burns; *27.* carcinoma; *28.* tumors of frontal lobe, chiasm and posterior fossa; *29.* alcohol (methyl); *30.* tabes; *31.* quinine; *32.* lesions of 3rd ventricle; *33.* abscess of brain and *34.* hyperthyroidism.

7. General symptoms are nausea; vomiting gastric irritability; respiratory disturbances; arthritis; polyuria; polydipsia; afternoon fever and increased pulse rate.

II. Objective Physical Findings: Because of the diversity and great variations of the subjective symptoms of suppuration of the sphenoid sinus, it must be remembered that a reliable diagnosis cannot be based upon symptoms alone, consequently objective physical findings play an important part in the diagnosis.

The rhinoscopic findings: There may be a septal deflection with secretions in the olfactory fissure noted after overcoming the turgescence. The pus appears either anteriorly between the middle turbinate and septum or posteriorly in the nasopharynx, occasionally with crusting on roof of nasopharynx located toward the choana. Often the oropharynx shows a dry, glistening appearance periodically streaked with pus; and while at times the secretion forms in crusts on the roof of the nasopharynx, it appears as fluid pus in the nasal cavity or olfactory fissure. When the drainage of pus is scanty, nasal suction may facilitate the detection of pus within the nose. In addition to the turgescence the inner side, and particularly the posterior tip of the middle turbinate, may show hypertrophy or hyperplasia, and if the case is of long standing the mucosa may assume a translucent appearance not unlike a beginning polyposis.

The presence of pus on the cotton of an applicator passed into the sphenoid fissure or into the sphenoid sinus demands further investigation. Nasopharyngoscopy is of much assistance although it may not always bring the osteum of the sphenoid into view, it frequently gives a fair view of the pus streaked olfactory fissure and sphenoethmoid recess. It demonstrates hypertrophied turbinates with their polypoid posterior tips, dilated blood vessel, granulations and edema about the sphenoid osteum.

The pharynx in addition to the periodic dryness and its pus streaked wall shows the typical granular and follicular pharyngitis with its enlarged or suppurating posterior pharyngical lymph nodes and scar tissue infiltration. The ears may reveal a catarrhal deafness noted by the functional tests and findings in the middle ears.

III. Radiography: The consensus of opinion is that radiographs of the sphenoid seldom show pathology. This opinion no doubt was brought about by the unsatisfactory radiography of the sphenoid sinus in the past and yet of some value, it led to erroneous conclusions. This was not only due to faulty technique and lack of standardization of positions but especially to the routine use of one position. It is my routine to require two positions, the Granger[35, 36] 107° angle (antero-posterior) and the Hirtz chin-vertex (vertical)[37], supplemented by the Rhese (oblique)[38] and Scheier (lateral)[39]. Radiopaque substances, such as lipiodol, are used as an auxiliary agent in radiology. It is of value in diagnosis of obscure conditions of the sphenoid, and in sphenoid suppuration is of value as it serves a two-fold purpose. It aids *1.* in determining the thickness of the lining membrane, and *2.* in the prognosis, as it is known that a long elimination time of lipiodol is indicative of a functionless membrane. Radio-

paque substances may be instilled into the sphenoid or may be used by the Proetz[40] displacement method.

The Granger position is the most universally used. In sphenoidal suppuration the "G" line becomes blurred or disappears.

The Hirtz position I have used some time as the position of choice for delineation of the sphenoid, particularly when a contrast medium is used. This position is also of value as it demonstrates extension of infection from the sphenoid to the base of the skull as shown by Pfahler[41]. With the head in the proper position the fluid level is clearly defined and with the aid of contrast medium by changing the position of the head it is possible virtually to reconstruct the cavity to be studied by means of exposures made in different planes. Because of the necessity of having a fluid level in making the Hirtz position it becomes necessary to have the patient in the sitting position with the head extended backward about 60°. In this position a fluid level will be seen in the sphenoid sinus, as well demonstrated by Baum[42]. When suspicious of an optic nerve involvement and radiography of the optic foramen are necessary, the Rhese position is of value as the foramen are clearly demonstrated within the orbits and their size and relation to the sphenoid are noted.

In the interpretation of radiographs the most essential point is the quality of the film. It is necessary that the film be of less density and less extreme contrast so that it may show the minute bone changes, the presence of finer changes in the soft tissue and the presence of fluid within the sphenoid.

IV. Exploration: Exploration or irrigation of the sphenoid sinus as a diagnostic aid has no equal in certainty and rapidity for determining the presence or absence of pus within the sinus. Irrigation with cytologic study during a remission is of much diagnostic and prognostic value. During acute infections and acute exacerbations of chronic infections, microscopy is unnecessary as the pus is demonstrative in the washings.

In many cases irrigation is done with little difficulty when the olfactory fissure is of sufficient width to permit the passage of the canula or trocar, but on the other hand greater diagnostic difficulties are encountered in cases in which the olfactory fissure is narrow and the anterior wall of the sphenoid and the osteum are not visible or a posterior ethmoidal cell overlaps the sphenoid.

Such a procedure is not free of dangers as penetration into the cranial cavity or hemorrhage may occur. Myles in 1882 made the statement that the sphenoid sinus was beyond the reach of human attack. Zuckerkandl first offered the suggestion that the sphenoid sinus could be penetrated by means of a trocar and since then

Schaeffer who first investigated the sphenoid sinus was followed by P. Watson Williams, Sieur, Jacobs, Menzel, Hagman, Lobell, Speilberg, Ingals and many others.

Investigation of the sphenoid sinus should be confined to such cases as present signs and symptoms which may be directly referable to the sphenoid. There are three groups of cases that require such investigation. Group 1 comprises such cases as manifest local and constitutional symptoms which could be traced to the sphenoid such as occipital headache, tinnitus, vertigo (sphenoid syndrome); pain back of eyes or over mastoid, mental symptoms, nasal obstruction, post nasal discharge, etc. Group 2, those patients who develop a sudden loss of vision with choked disc (optic neuritis) in which no cause has been found. Group 3, patients suffering with intracranial complications as meningitis, brain abscess, and general sepsis of undetermined origin[43].

The cases which comprise Group 1 require differential diagnosis as they may be easily confused with posterior ethmoid disease.

The demonstration of suppuration in the olfactory fissure is a finding which indicates a disease of the sphenoid but is no absolute proof of its existence as the objective symptoms are also somewhat characteristic of suppuration of the posterior ethmoidal labyrinth, therefore, the two conditions should be considered together in the diagnosis until one has definite proof that the origin of the secretion is from the sphenoid sinus and until then it is impossible to establish the diagnosis with certainty. This proof consists *1.* in the direct observation of the purulent drainage from the osteum of the sphenoid sinus, and *2.* in the demonstration of secretion from the sphenoid sinus by means of irrigation.

After a true suppuration of the sphenoid exists the question arises, is this the only source of suppuration or is the sphenoid disease combined with an involvement of the posterior ethmoid labyrinth[44]? After irrigation of a pus containing sphenoid sinus, the reappearance of pus in the olfactory fissure proves its origin in the ethmoid labyrinth. Also the observation of pus draining from the ostea of the ethmoid by means of the nasopharyngoscope proves a combined ethmoid infection.

Often the controversy arises as to whether the empyemia is the result of sphenoidal suppuration or pyosinus. Regardless of whether it is or not, it has little effect upon treatment. Group 2 and 3 comprise cases in which the optic nerve is involved or orbital or intracranial complications are impending. In such cases one must agree with Lynch, Beck, Herd, Eagleton and others that conservative treament gives nothing more than passing help[45], [46].

B. Conservative Treatment: Conservatism, however, is an entirely relative term and its implications vary at different times. In this discussion it implies any management of this disease save surgery upon the sphenoid or other sinuses. The conservative or nonsurgical treatment of sphenoid suppuration must be resorted to in properly selected cases for its successful application, and under no circumstances should anyone believe the nonsurgical methods a panacea, nor attempt to make them replace appropriate surgery in cases frankly surgical. Sphenoid surgery will ever play an important place in the practice of rhinology but as the nonsurgical methods are better understood they will be given more and more consideration and possibly in the future supplant some of the surgery now in vogue[47].

Although conservative treatment comprises constitutional and local management, it would not be proper to discuss conservative treatment without at least mentioning prevention of the disease.

I. Prophylaxis: Today scientific medicine is more and more concerned with the consideration of the prevention of disease, consequently if the opportunity be given prevention of sphenoid infection should be practiced. The prophylaxis of sphenoid suppuration calls for, *1.* prophylaxis of acute upper respiratory infection. *2.* General hygiene. The prevention and correction of physical causes as, lack of exercise, improper clothing, chilling of body, loss of sleep, dieting, excessive perspiration with exposure, overheating, bathing, draughts, constipation, etc. *3.* Nasal and oral hygiene. The proper education of children as to how to properly blow the nose and cleanse postnasal space, to avoid nasal irrigations, to properly and regularly brush the teeth, and cleanse the pharynx. *4.* Predisposing factors such as allergy, mechanical nasal obstructions to ventilation and drainage as septal deflections, neoplasms, hyperplastic turbinates and foreign bodies of the nose, should be properly handled. *5.* Diet should be regulated especially in regard to vitamines A and D. *6.* The endocrines should be studied and any dyscrasia corrected. *7.* All local respiratory infectious causes should be properly cared for as diseased tonsils, adenoids, teeth and gums. *8.* Plenty of sunlight. *9.* Remove the extrinsic and intrinsic factors which may cause or perpetuate a respiratory infection as exposure to heat, cold and excessive humidity in house or office and to avoid gases and vapors by poor ventilation.

II. General Treatment: Fortunately these cases usually respond readily to general and local management. When the condition occurs as a complication of some systemic disease (e.g., influenza, scarlet fever, etc.), the general treatment must obviously include the treat-

ment of that disease. The marked tendency to recurrence even after constructive surgery[45] (submucous, resection, turbinectomy, infraction of turbinates, polypectomy, tonsillectomy, adenoidectomy, and teeth extraction), seems to prove a constitutional basis in many cases. Internal or constitutional remedies should therefore play some part in their management. The treatment in acute cases should begin with rest in bed and a thorough cleansing of the alimentary tract. In the chronic cases presenting evidences of lowered vitality or resistance constructive tonics should be prescribed, and in the overfed, full-blooded or plethoric case careful regulation of diet, restriction of alcoholic drinks and regulation of bowels should be advocated. In all cases, smoking should be discouraged. Suitable regular exercise should be recommended, diet should be regulated so that patient receives sufficient vitamines and plenty of fluids with alkalinization[49, 50]. Atropine in small doses frequently administered by mouth is an aid in acute cases in checking glandular activity. Iodides, urotropine and salicylates have been of questionable value. Occassionally when sphenoid suppuration is a part of a panisinusitis the result of a metabolic upset incident to heart, kidney or gastrointestinal diseases, it becomes necessary to treat these conditions. When the background is specific such as lues, tuberculosis, diabetes, etc., insist upon specific treatment in conjunction with the other methods of therapy. Proteid therapy (milk omnadin, aosan, caseosan, novoprotein, phylogetan, terpichin, caseal, lactosen, yatren, casein, stomasin, alfasol, tuberculin, and typhoid bacillus), bacteriophage, vaccines and leucocytic extract (Hiss), have proven of little value, although Herelle[51] has reported good results with bacteriophage in other infectious diseases and it would be interesting to know its results in sphenoid disease.

III. Symptomatic Treatment: Symptomatic treatment consists in the relief of pain and headaches by means of anodynes, narcotics, analgesics, physio-therapy and electro-therapy. Cocainization of the spheno-palatine nerve or injection of the nasal ganglion is recommended in those cases where neuralgia is an aggravating symptom not relieved by anodynes and in patients who resent surgical intervention. The restoration of nasal ventilation and the favoring of the discharge of secretions will be discussed under local treatment.

IV. Local Treatment: In regard to local treatment the reestablishment of drainage and ventilation is the objective.

1. Shrinking Solutions: As cocaine, metaphedrin, ephedrin, adrenalin, adrephin, etc., have been used. The use of these drugs for shrinking the turbinates is so generally known that extensive discussion would not add to their recognized merits[52]. They may be

used in the anterior nares or when enlarged turbinates or deviated septums make it impossible for the solution to reach the diseased area, the oral or nasopharyngeal route suggested by Pressman[53] is preferable.

2. Suction: It may be used in cleansing the meatuses of the nose in place of irigation and for draining the sinus when the head is bent forward with the osteum dependent.

3. Packs: The packing of the nose with various silver preparations (argyrol, silvol, collens, ichthyol and glycerin) and dyes as recommended by Dowling and Skillern has not given me the results that others have claimed for them.

4. Lavage: Lavage or irrigation of the sphenoid in addition to being of diagnostic importance is of great therapeutic value. It is surprising how the removal of a small mass of pus by lavage will give marked relief of symptoms. Lavage may be done as Lobell[57] has described by means of the direct (through osteum) or indirect (by puncturing its anterior wall with a needle and forcing the return flow through the osteum) method. An indirect puncture of the anterior wall made after radiographing the patient with the needle in place facilitates the procedure and makes it an accurate and safe procedure. It is the method of choice in cases where the ostea are small and displaced or the pus very thick and inspissated. Lavage at times is impossible because of nasal obstruction. Many canula and needles have been used for this purpose[58, 59].

5. Displacement irrigation as recommended by Proetz[60] is of some value. Many drugs have been used but normal saline has been my choice. The important factor in this type of therapy is the dilution of the pus which facilitates removal, although at times it is difficult or impossible to have the fluid enter the sinus.

6. Diathermy: Submucous electro coagulation (Beck)[61] of the turbinate overcomes the turgescence and shrinks the hypertrophied or hyperplastic turbinate.

7. Radium as an adjunct in cases associated with ethmoidal involvement where polyps may interfere with drainage and ventilation of the sphenoid is of value.

8. Ultra-Violet irradiations of the nasal ganglion has been advocated by Odoneal[62] for the relief of pain.

9. Steam Inhalations often aid in shrinking the nose and are cooling and refreshing to the nasal cavity and throat and give relief of tracheal irritation in acute cases.

10. Heat applied to the side and back of neck has been recommended for the relief of occipital and mastoid pain.

11. Intubation with silk retention catheter was discarded because of its irritation to nasal mucosa.

12. Powder Insufflations of iodin, boric acid, etc. is found to be irritating and are not the most promising.

13. Instillation of drugs as antiseptics and ferments have been discouraging and the use of oils, jellies and ointments in the sinus or nasal cavity are not theoretically correct from a pharmacodynamic view point[63].

14. Ionization of the sphenoid was used in our clinic as early as 1925 and discarded, nevertheless Campbell found it in some cases especially useful[64, 65].

15. Besredka Antivirus[66] has had very little trial although in my limited experience with it, it offers sufficient encouragement to warrant further investigation. Of 8 cases of sphenoidal suppuration treated, 6 were treated by instillation and are free from purulency and symptomatically well for a period of 1 to 11 months. The other 2 cases were treated by Proetz displacement method using the antivirus, and both apparently well. None of the above 8 cases required surgery. Recently I have been using Abbott's staphylo. jel.

C. Conclusions: That treatment of this condition has proved a troublesome problem in rhinology is shown by the long list of unrelated therapeutic measure which have been proposed for its relief, and in addition to what has been proposed I should like to suggest that research work be carried out with Besredka antivirus and bacteriophage. And in conclusion I wish to state that although this subject is not conclusive, if anyone has been stimulated by this paper with a new impetus to investigate in a research way antivirus and bacteriophagy or to make a study along the trend of biochemistry, I feel that the presentation of this subject has been well warranted.

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914 American Nat'l. Bank Bldg.

ACUTE ABSCESS IN THE BODY OF THE SPHENOID, RUPTURED INTO THE PITUITARY FOSSA: AUTOPSY REPORT.

DR. ALTON L. GRANT, JR., Auburn, Me.

Acute abscess in the body of the sphenoid is extremely rare. Careful perusal of the literature on the sphenoid reveals little. There was nothing in the history and symptomatology in this case which would point directly to involvement in the sphenoid. The entire clinical picture could be easily accounted for by pathology elsewhere which was not the primary cause of death as found at the autopsy.

Skillern[1] states: "It is rare that the sphenoid becomes acutely infected *per se* without some of the other accessory sinuses, particularly the posterior ethmoid cells, sharing the infection. The latter, however, by reason of their better drainage may entirely recover, leaving the disease isolated in the mucosa of the sphenoid. This is particularly true in those cases which follow the infectious diseases, notably influenza." He further points out that, "from a rhinological point of view the diagnosis of acute empyema is seldom made. The general symptoms, at least at the commencement of the attack, furnish no clue pointing toward any particular sinus, as they are identical with, or perhaps only a slight exaggeration of those commonly associated with ordinary acute coryza."

Mrs. X, age 40 years, admitted to the Central Maine General Hospital, Lewiston, Me., May 1, 1931, at 10 a. m. Patient in a semicomatose condition, temperature 105.5°, pulse 100, respiration 22.

Family History: Irrelevant. *Past History:* Her son claims that she always enjoyed good health, was subject to occasional head colds. Appendectomy performed several years ago. Had two children, both normal births. Never suffered from sinus trouble or any so-called "nasal catarrh."

Present Illness: Six days before admittance to hospital she was enjoying her usual good health. On Monday of the same week her illness was ushered in with a severe head cold. This was soon followed by terrific, continuous, generalized headache; a little worse, if any, in the occipital region and more severe at night. On Wednesday both ears ruptured and discharged freely. Her severe headache persisted all the time. She obtained no relief from the ordinary headache remedies. Early Friday morning she became stuporous. The family physician was called and he immediately sent her to the hospital.

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Physical Examination: Patient well nourished, head retracted and neck rigid. Pupils round, equal in size and react to light. Fundi occuli negative. Both ear drums ruptured, red, patulous and discharging freely. Discharge serosanguineous in character. No redness, swelling or tenderness on pressure over either mastoid. Nose and throat essentially negative. Blood pressure 140-90. *Heart and Lungs:* Within normal limits. *Abdomen:* Soft. No distention, no tenderness. *Reflexes:* Knee jerks increased; slight ankle clonus, Kernig's sign positive. *Lumbar Puncture:* Spinal fluid very cloudy. Pressure greatly increased. Cell count, 6,500 polys. A Gram positive diplococcus was present.

Clinical Progress: Her condition grew rapidly worse. She went into a deep coma. About three hours before death, her temperature soared to 109.5°. Death took place about 48 hours after admission to hospital.

Clinical Diagnosis: Acute suppurative otitis media. Diffuse meningitis.

AUTOPSY REPORT[2].

The body is that of an obese white woman of about 50 years, showing complete rigor mortis with moderate lividity. Hair streaked gray and abundant. Sclerae clear; pupils, 0.4 c.m., rounded and equal; both lower eyelids and loose subjacent tissue present a purplish discoloration with a moderate degree of pitting edema. Dependent portions are flat to percussion. Abdomen soft and not distended.

Scalp strips readily. Calvarium presents no structural changes. On its removal, dura is firmly adherent and from which it is separated with considerable difficulty. Brain substance bulges; vessels of pia-arachnoid are well injected; brain substance is moist and edematous; vessels throughout are congested; pia-arachnoid smooth and lustrous throughout except at the base of brain. Portion enclosed by circle of Willis shows a dense and fibrinous exudate. A less dense deposit is present over the pons, temporal and frontal lobes. Temporal and frontal lobes are slightly agglutinated by a fibrinous exudate. On section, ventricles appear slightly distended and are occupied by a turbid fluid. Choroid plexus on either side are injected and appear matted together. Except for prominence of vessels throughout and a definite increase in moisture, brain substance shows no other structural changes.

Hypophesis is retained *in situ* upon removal of the brain and is dissected with slight difficulty; appears edematous and injected. Cella turcica presents a brownish appearance, is markedly softened, admits a chisel with slight resistance. A considerable amount of purulent exudate escapes upon exploration. Entire body of sphenoid

is similarly involved. Wings of sphenoid present no structural changes. Other osseous structures, mastoids, tegmen tympani, petrous pyramids, maxillary sinuses and ethmoids are not remarkable. Weight of brain, 1425 grams. Detailed description of the abdominal and thoracic contents will be omitted. Pathological findings will be included in the summary.

Microscopic Findings: Heart: Shows slight fatty degeneration and fatty infiltration. *Lungs:* Shows alveoli of indurated areas packed with leukocytes, the majority of which are polymorphonuclear in type. These are inmeshed in fibrin. Leukocytes stain well. Bronchus and bronchioles show a moderate mono and polymorphonuclear infiltration. *Liver:* A moderate degree of fatty degeneration with central veins engorged. *Spleen:* Germinal centers show frequent mitotic figures. Marked degree of hyperplasia is present. *Kidneys:* Glomeruli are freely infiltrated by polymorphonuclear cells and show an increase in vascularity. Lining cells of tubules are frequently granular and edematous and in many situations stained poorly. *Sphenoid:* Sections taken from body of sphenoid show large collections of polymorphonuclear cells and the presence of numerous Gram positive diplococci. *Brain:* Shows vessels markedly distended with a marked degree of edema. Sections elsewhere are not remarkable.

PATHOLOGICAL DIAGNOSIS:

1. Abscess of body of sphenoid. *1A*. Acute osteomyelitis of sphenoid. *2*. Basilar meningitis. *3*. Cerebral edema. *4*. Acute otitis media. *5*. Spinal meningitis. *6*. Bronchopneumonia. *7*. Chronic myocarditis. *8*. Acute splenic tumor. *9*. Acute glomerular nephritis. *10*. Toxic nephrosis. *11*. Passive congestion of liver, spleen and lungs.

The outstanding features of this case appear to be:

1. The short duration of the entire process.

2. The virulence of the infection.

3. The importance of terrific headache following a so-called head cold.

4. The double otitis media, which proved to be misleading and was thought to be the primary cause of the meningitis.

5. It is probable that this involvement in the sphenoid was an acute exacerbation of an old latent infection which was the result of an extension from some previous posterior ethmoid infection which had resolved. Pathology gross or miscroscopic in the other sinuses could not be demonstrated.

REFERENCE.

1. SKILLERN: Accessory Sinuses of the Nose, p.p. 378-379-380.

2. Autopsy performed by Dr. Julius Gottlieb, Pathologist, Central Maine General Hospital, Lewiston, Maine.

133 Court Street.

AN UNUSUAL FORM OF ACUTE FRONTAL SINUSITIS.

Dr. I. Ruebert Smith, Toronto.

Patient was referred to me by Dr. A. Brodey, complaining of pain over her right frontal on December 20, 1929, with a history of three days' duration.

Examination showed the nasal mucosa in right nasal passage congested with discharge in the upper part of the passage. On shrinking the upper part of the right nasal passage a small quantity of creamy discharge was drawn away which gave her relief for a few days.

Radiograph taken on December 21, at the Toronto General Hospital showed the following report (see Fig. 1):

"The frontal sinuses are well visualized. Here the frontal sinuses are well seen, and no definite evidence of any change on the left side can be detected. On the right side a very minute degree of thickening is present in the Water's position."

On December 24, she returned with pain just as severe as in the previous attack. Radiograph taken (see Fig. 2), with report as follows:

"Comparing the frontal sinus on the right side at this examination with the previous plates, we could feel that there was a definite increase in cloudiness here, which, in our opinion, would indicate an empyema in the right frontal."

Patient treated in the office on December 26, 27 and 28, with slight improvement. On December 29, she complained of severe pain over right frontal. Admitted to the Western Hospital on December 29.

Radiographs were taken of frontals on January 1 (see Fig. 3), which showed some clearing of the right frontal, but a circumscribed subperiostial swelling on the left side of septum, with a slight dehiscence in the septum between the frontals.

Patient was running a slight temperature, 99°, with pain still localized over her frontals; pain in back of head. Complained of drowsiness, with occasional relief of pain; fair nights. This condition lasted for a few days and was followed by pain in her chest and right shoulder.

Dr. George Young, consulting physician, was called in and found nothing wrong intracranially or in the chest, but thought the patient was of an extreme neurotic type.

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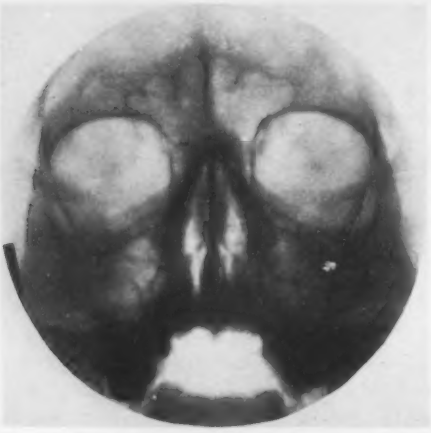

Fig. 1. Taken December 21st. Frontal sinuses are well visualized. No definite evidence of any change on the left side can be detected. On the right side a very minute degree of thickening is present.

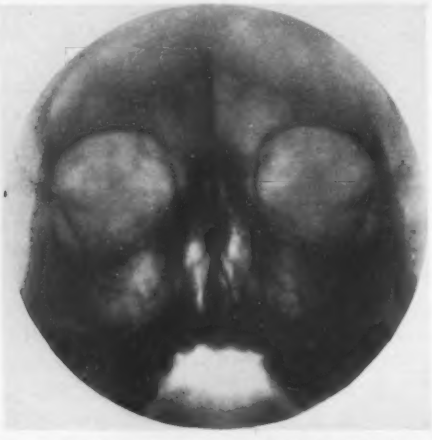

Fig. 2. Taken December 24th. Comparing this with plate No. 1 you will observe a definite increase in cloudiness in the right frontal.

On January 6, X-ray radiograph (see Fig. 4) was taken of frontal sinus, which showed slight diminution of the subperiostial swelling in the left frontal sinus.

During the week she complained of indefinite pains in head, chest and shoulder. No rise of temperature; fair nights.

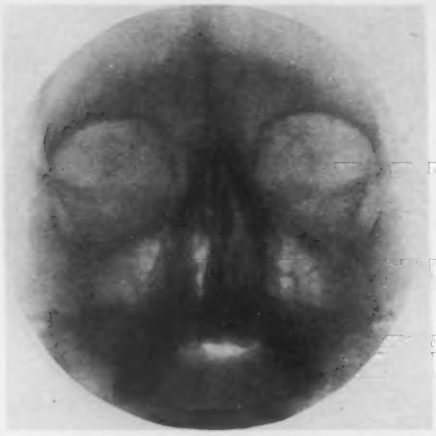

Plate No. 3. Taken January 1st. Note some clearing of the right frontal with a circumscribed subperiostial swelling on the left side and some dehiscence in the septum between the two frontals.

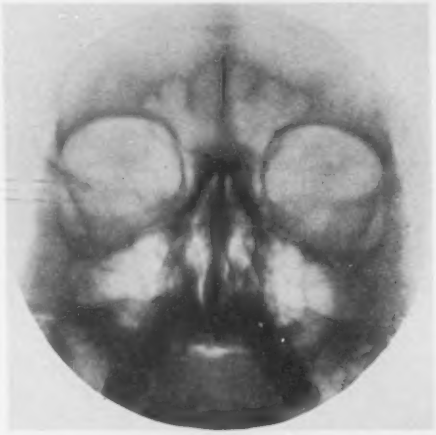

Plate No. 4. Taken January 6th. Compare the subperiostial swelling here with that taken in previous plate and you will observe there is a slight diminution in its size.

On January 12, profuse purulent discharge came away from the left nasal passage, with relief of her frontal headaches.

On January 13, first complained of pain in her right iliac region, also pain in right groin, with nausea and vomiting. No rise of temperature.

Dr. Frank Scott, general surgeon, was called in. Ordered kidney and bladder X-ray. The following report was returned:

"The kidney shadows are not unusually large, or low. We do not find any definite indication of calculus within either kidney, the path of either ureter or within the bladder area."

A diagnosis of appendicitis was made.

On January 19, radiograph of frontal sinuses made (see Fig. 5). You will notice how the subperiostial swelling completely disappeared. Both frontal sinuses appear clear.

January 22, appendectomy performed. Following is the pathologist's findings:

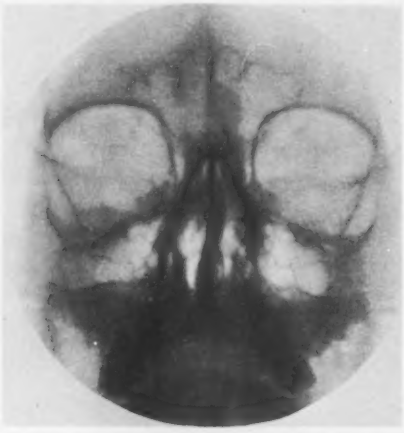

Plate No. 5. Taken January 19th. Notice the disappearance of the subperiostial swelling and clearness of both frontals.

"*Macroscopic Findings:* This measured 6 cm. in length, is normal in size, pale and firm under pressure. Gross section shows considerable thickening of the wall on one side. The lumen is narrowed and contains a slight amount of bloodlike serum.

"*Miscroscopic Examination:* There is a chronic inflammatory change of the peritonial coat which shows some hyaline degeneration. The wall is enormously thickened and scarred and the submucosa is thickened and shows some fat vacuoles. The lymph follicles are slightly swollen. There are no pus or eosinophiles about the mucosa.

"*Diagnosis:* Chronic appendicitis."

Patient made an uneventful recovery.

During the period of her illness, blood counts were made on several occasions, which showed no change in the red cells with a leukocytosis which varied between 8000 and 9000.

Reports on various urine examinations were negative.

Conclusion: An unusual form of frontal sinusitis which went through an unusual course with complete resolution without operative interference, but apparently being the exciting etiological factor in an acute attack of chronic appendicitis.

116 College Street.

BRONCHOSCOPIC AND ESOPHAGOSCOPIC DIATHERMY ELECTRODES.

DR. M. C. MYERSON, New York

Two electrodes for use through the bronchoscope and esophagoscope are offered. The electrodes are made with straight and right-angled ends.

The straight-ended electrode (A) was first designed and used by Dr. Arrowsmith for fulguration purposes several years ago and it is the writer's purpose in this communication to call the attention of

the profession to it. The second electrode (B) has a right-angle tip which enables the operator to see the end of the instrument while he is using it in the bronchus or esophagus.

The electrodes are made in suitable lengths for use through the average bronchoscope and esophagoscope. They are of value for desiccating or coagulating a carcinoma of the bronchus or esophagus and have proven useful in reducing the size of bronchial adenomata.

12 East 86th Street.

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THE RELATIONS OF DENTAL INFECTIONS TO MAXILLARY SINUS DISEASE.*

DR. VICTOR R. ALFARO, Washington.

General Considerations: It has been known for a long time that dental etiological factors, either infectious or traumatic, are very common in diseases of the antrum of Highmore. Just what percentage of all maxillary infections can be attributed to the dental origin is still a matter of dispute. Some of the early authors gave it as high as 90 per cent, while some more recent investigators only give it 8 per cent. But perhaps the pendulum has swung too far, so that not enough consideration is being given to dental pathology today. That a careful transillumination of the teeth should be included in the routine examination of the rhinologist has been strongly advocated. It is surprising how many times one is able to pick up diseased teeth that may have gone unnoticed by the patient or by a careless dentist. The rhinologist should co-operate heartily with the surgeon-dentist in the treatment of this closely allied field. Undoubtedly many failures are due to a not too thorough investigation of possible dental causes in antrum disease. There is an intimate anatomical relationship between the roots of the upper teeth and the floor of the maxillary sinus. Due to the variations in shape and development of the antrum, we may have all the molars, premolars and cuspids in close contact with the antral floor, or, on the other hand, the thickness of the alveolus may be so great that none or only some of these teeth may be in contact with the floor. Those most commonly found in close relationship to the antrum are the second premolar and first molar.

Etiology and Pathology: The dental causes of antrum disease present themselves in two large groups. First, purely pathological; secondly, traumatic.

1. Among the purely pathological causes we have first, in order of occurrence, abscesses of the teeth. These may further be classified into apical and parietal, the first being found at the root or roots of the teeth and the second around the body of the teeth. They may be either diffuse or circumscribed, as in the case of the granuloma. Again, in regards to their relative position to the antrum floor, they may be considered as being either adjacent or remote. The adjacent

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abscess is right under the antral floor and may be separated from it by bone of varying thickness, or covered by mucoperiosteum alone. This condition constitutes what Novitsky calls the antrum drain[1]. The remote abscess may be anywhere in the superior maxilla or in the mandible, where it may drain into the inferior dental canal[1]. The pathology of a dental abscess is that of any bone abscess; there is always necrosis and resorption of bone tissue. A diffuse abscess is not properly walled off and this is the type that more commonly finds an entrance into the maxillary floor by continuity of tissue. The circumscribed abscess or granuloma is properly walled off and may be extracted completely with the tooth.

It is perhaps wise to bring in here a consideration of that dental bugbear, the pulpless tooth, since so many dental authors are at variance in regards to this subject. As we understand it, the pulpless or devitalized tooth is one in which the nerve in the pulp has been destroyed or the blood supply so impaired that there is a necrosis of the soft tissues in the dental canal. The radical oral surgeon advocates the extraction of all pulpless teeth, even if they are radiographically negative. In this respect Mead believes that the most dangerous spot where a devitalized tooth can be found is in close proximity to the floor of the maxillary antrum, because as he explains, when there is disease at the apex of the tooth, but no bone to be absorbed, it may be negative radiographically, but actually a dangerous focus of infection that will eventually invade the maxillary sinus. The tremendous importance of adjacent abscesses to antritis is, therefore, evident; but the possibility should not be overlooked of remote abscesses, say in the mandible, infecting, by way of the blood stream, any of the paranasal sinuses. This probably occurs more frequently than it is suspected, particularly when there has been previous pathology of the sinuses, with a commensurate lowering of their powers of resistance. The antrum then becomes a *locus minoris resistantia* that lights up sporadically because of a dental focus of infection, or, for that matter, any focus of infection.

Second in importance to abscesses is periostitis. This may be primary, but perhaps more commonly secondary to an abscess. The infection, reaching the periosteum at the floor of the antrum, may spread along its surface and cause a rupture and subsequent infection of the antral mucous membrane. These infections are usually severe; they may terminate simply as a mucoperiostitis, or, on the other hand, may involve a greater or lesser part of the alveolar bone.

The third etiological factor is osteitis, or, as there seems to be some confusion in the terminology of different authors, an osteo-

myelitis. Whether this infection is primary or secondary, or both, is of little interest to us, but when it occurs a small or a very large part of the mandible and antral floor may be involved. The bone necrosis is then the causative factor of a chronic empyema of the antrum. The discharge is always extremely fetid. The mucous membrane of the antral floor may be polypoid or necrotic, and the whole membrane may at times appear gangrenous. These infections are the most severe and the hardest to treat, but, fortunately, quite rare. Another etiological factor of maxillary sinusitis, but perhaps the rarest, is the dentigerous cyst. Cysts may be inflammatory or follicular. The first type grows from a granuloma and advances by a pressure necrosis pointing either in the canine fossa or within the maxillary sinus. Occasionally in punctures of the antrum, one of these cysts may be entered and a clear yellow fluid recovered. The follicular

Fig. 1. These teeth extracted from an osteomyelitic jaw illustrate how large openings are made occasionally into the antrum.

cyst develops from unerupted or supernumerary teeth. Due to their rarity, they are considered as only a small part of antrum disease.

Another etiological factor, which is less recognized in antritis, is periodontia, commonly known as pyorrhea. This disease, which brings about a destruction of the alveolar border with loosening of the teeth, may infect the antrum by continuity if extensive enough, or through the lymph or blood stream if the area is small and circumscribed.

Tumors do not of their very nature come within the scope of this paper, though any tumor may have a dental origin and eventually invade the antrum.

2: A very large group of dental born maxillary disease is due to dental trauma of one type or another. One must not be too quick in condemning the surgeon-dentist who occasionally invades the antrum in the extraction of teeth or following extraction by

curetting an abscess cavity. As mentioned before in this paper, the teeth most commonly found to communicate with the antrum are the second premolar and first molar. Though some of the dental accidents may be due to the carelessness of the operator, it can truthfully be said that they are decidedly in the minority. The unavoidable trauma subsequent to the extraction of an impacted third molar near the posterior edge of the sinus, or the extraction of residual roots anywhere along the floor, or curetting of an abscess cavity may all produce severe antral empyema. On the other hand, a great proportion of cases, in which the antral floor has been perforated, go on to uneventful recovery, because a healthy sinus has been able to overcome the insult. As Mithoefer[2] points out, there

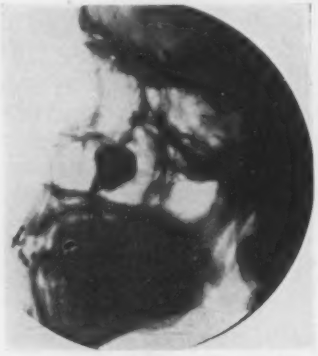

Fig. 2

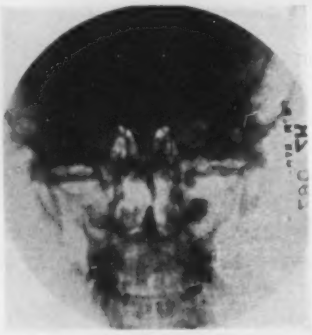

Fig. 3

Fig. 2. Lipiodol injection of the antrum through a chronic alveolar fistula. Note the filling defect of the antrum due to inflammation of the mucosa.

Fig. 3. An impacted third molar pushed into the antrum when extraction was attempted. This radiograph was taken as soon as the accident happened and shows a clear sinus. A virulent empyema, however, developed. The tooth was removed later when Caldwell-Luc was performed.

is a certain proportion of cases with latent maxillary disease which may light up as soon as the local or general resistance of the patient is lowered. Any dental trauma lowers considerably the resistance of the antrum which will light up subsequent to an extraction producing a severe empyema. The patient is very prone to blame the dentist, when as a matter of fact the disease was present all the time and was only brought acutely to his attention at the expense of the dentist. The rhinologist should, in their behalf, explain these things to the patient and thuswise save many unpleasantries. It is not a

rare accident for a dentist in removing a difficultly placed impacted molar to fracture a root within the antrum, or, what is more serious, to force the whole molar into the antrum. We saw one case in which an impacted third molar was pushed clearly through the antrum floor, only remaining covered by the mucoperiosteum. The patient developed a very virulent empyema and when the acuteness subsided a Caldwell-Luc was performed and the tooth removed with great difficulty. There was some necrosis of the bone surrounding the tooth, which was curetted at the time by the dentist. In a series of Caldwell-Lucs studied with Trible it was found that out of 39 cases, 17, or 43 per cent, had a definite relation to a dental etiological factor. The most common was traumatism following extraction or other dental surgery; and these were by far the most severe cases and the most difficult to cure. A comparison of Trible's figures shows

Fig. 4. Case of osteomyelitis of the maxilla of dental origin; chronic empyema of the antrum. A Caldwell-Luc was performed but healed with difficulty, leaving two external fistulae and one at the site of incision in the canine fossa.

clearly that a greater percentage of dental born infections require radical surgery than antritis of nondental origin.

3: Retrograde Infections: For the same anatomical and pathological reasons that the teeth can infect the antrum, the antrum may infect the teeth. It is, therefore, hard to decide which the primary factor is in infection of teeth and sinus. Skillern[8] reports a case in which, without room for doubt, perfectly healthy teeth were infected following antral disease. One, therefore, has to contend at times with a vicious circle and a thorough eradication of all infected parts is necessary.

The symptoms of maxillary sinusitis of dental origin are the same as those from other causes, though perhaps usually more severe. Patients with negative sinus findings, exhibiting symptoms of vague pains in the maxilla and alveolus extending over the frontal and

temporal areas, should not be regarded as neurotic, without a thorough search for possible dental pathology.

Treatment: The treatment of these conditions is at first the conservative treatment of any antritis; if dental pathology is found, it should be attended to immediately by a competent oral surgeon, with a view of removing any necrotic bone or granuloma. Cases referred to the rhinologist by a dentist who has perforated the antral floor should be treated in close co-operation with the dentist in the following manner: If it is evident that there was no disease present in the antrum at the time it was perforated, a careful suturing of the gums should be immediately advised. As said previously, many of these cases heal spontaneously and never require either dental or intranasal treatment. The patient should be sent to the rhinologist for observation and, if necessary, a radiograph of the affected sinus is taken. This has been found of unquestionable value. If at the time the antrum was perforated the dentist discovered the presence of pus, indicating clearly sinus disease, which the patient may or may not have known existed, he should close the dental wound and refer the patient to the rhinologist for immediate intranasal treatment, needle punctures, lavage, etc. Many cases will respond quickly to these conservative procedures. Sometimes, however, a large window in the nasoantral wall under the inferior turbinate will be necessary to effect a cure. If the dental trauma has been so severe or the resistance of the patient so low that a periostitis or osteomyelitis of the maxilla and antral floor develops, the treatment will have to be more radical. Just what the surgical approach should be is governed by the individual case. We firmly believe that the operation should be conducted by the rhinologist and the oral surgeon together. The usual procedure is for the oral surgeon to remove infected teeth, and curette necrotic bone first. If, as happens in some few cases, the oral surgeon has been compelled to remove a large part of the antral floor before he found good bone, a good view of the maxillary antrum results. The rhinologist can then work in the antrum easily, without having to sacrifice its canine wall. If an intranasal opening has not been made before, it should be made at this time. When the antrum has been thoroughly cleaned it is packed with gauze through the alveolus and brought out through the intranasal opening, just as in the Caldwell-Luc. The two flaps of the gum, with which as much of the periosteum as possible has been preserved, are then sutured tightly by the oral surgeon. If, on the other hand, and this is the more common case, only a small opening is made in the antral floor, the rhinologist's approach should be, as in the Caldwell-Luc, through the canine fossa. The dental wound should be tightly sutured

and the rest of the operation and postoperative treatment belongs entirely to the rhinologist. Some of these cases with large alveolar openings occasionally break down and form a fistula; they are extremely hard to cure. For this reason the alveolar approach has been emphatically condemned as one of choice. It should only be one of necessity. The danger should also be stressed of treating the antrum conservatively through an alveolar opening because of the frequent formation of permanent fistulas. Many dentists who treat antritis by irrigations and packing of an alveolar wound are incurring the aforementioned danger. It is true some cases will get well when treated this way in spite of the treatment, but that does not justify the procedure. It stands to reason that the infection will seek to drain by its lowest point. If this alveolar opening is closed tightly as soon as it is made and an intranasal opening established, the empyema will drain intranasally and relieve a healing alveolar perforation of an extra burden.

CONCLUSIONS.

1. Many infections of the maxillary sinus are due to frank or hidden dental disease or to unavoidable dental trauma.

2. A careful transillumination of all the teeth should be a routine procedure with every rhinologist, supplemented by a dental examination in suspicious cases.

3. A careful examination by the dentist should be advised in all cases of maxillary sinusitis that do not respond to treatment in a reasonable length of time.

4. The radical surgical treatment of chronic antrodental pathology should be performed by the dental surgeon and rhinologist together. A close co-operation with the dental surgeon at all times will decidedly reduce the percentage of failures.

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1801 I Street, N.W.

AN AURAL PROTECTOR AGAINST NOISE AND WATER.

DR. ARTHUR C. JACOBSON, Brooklyn, N. Y.

Two purposes are served by the device described and illustrated in this article: the total exclusion of water from the ears, and the partial exclusion of sound.

The first purpose is desirable for protection of the ears while swimming, diving or bathing (shower and surf). It is not alone the ears with perforative and other lesions that need to be protected in the water; all ears should be safeguarded.

The second purpose is desirable in the following circumstances: whenever noises unduly inhibit or prevent sleep, disturb the nervous system to an injurious extent, promote deafness, interfere with mental concentration, annoy sensitive patients or lessen industrial efficiency. Very special circumstances will suggest themselves, *e. g.*, aviation, cannonading, preoperative and postoperative "insulation" against sounds in thyroid surgery, scopolamin-morphin anesthesia (in place of the oiled cotton now used), preoperative preparation with the newer agents, the use of hypnotic drugs, and the relief of some cases of tinnitus, etc.

The device is extremely simple in principle and construction, inexpensive, of adjustable tension, easily fitted to practically any head (three sizes suffice for most), light in weight (5 drams), sterilizable by boiling, and does not enter any part of the auditory canal

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but merely closes it by "shutting the door" (the tragus). The light spring can be easily bent with the fingers and the molded hemispheres of sponge rubber, which close the ears, are notably resilient, thus applying pressure gently.

Diving does not displace the device. In swimming by means of the modern strokes, movements of the temporomandibular articulation occasioned by the opening and closing of the mouth in breathing do not displace the protector, which makes the necessary surface adjustments automatically.

The common noises are all markedly reduced in intensity, depending, of course, upon distance and character; so much so as to be largely deprived of their disturbing or injurious effects upon the nervous system.

Fig. 1. Aural protector.

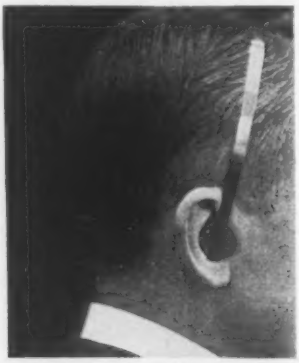

Fig. 2. Aural protector in use.

Elaborate recommendations are made by our noise commissions but nothing is ever said about the obvious need: artificial inhibition or suspension of the hearing faculty itself when under stress. The noises incidental to this machine age cannot be wholly abated, much less abolished. We must, therefore, use all our ingenuity in circumventing them. Man as we know him represents a survival attributable to a long series of biologic adaptations and adjustments; now it is up to him to use his wits consciously to protect himself against anything tending increasingly to handicap him, even to the point of taking advantage of adverse circumstances to augment his resources. As in the case of the other special faculties, resting of the sense of hearing, when under undue strain or when it is fatigued or impaired, tends to conserve and enhance functional integrity.

24 Clinton Street.

International Digest of Current Otolaryngology.

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Dr. H. C. Rosenberger, Cleveland.

H. W. Barber, in the Proceedings of the Royal Society of Medicine, Section on Otology, August, 1931, presents a detailed paper on "Eruptions Involving the External Auditory Meatus." The author is a dermatologist who has made a careful study of the auricle and, as such, is in a position to more carefully and scientifically discuss skin affections in or about the pinna. He classifies the eruptions as due to external infections; as due to infected organisms reaching the skin through the blood stream; as due to allergy; as due to involvement of cutaneous nerves, and conditions not included in the above list. This paper may well be read and studied by practicing otologists.

Harkavy and Maisel, of New York, in the *Jour. A. M. A.*, May 30, 1931, write on Infectious Asthma and Its Relationship to Chronic Sinus and Pulmonary Disease. They state that allergy may be clouding the physician's judgment and hindering him from a true conception of the pathology in this type of case. They claim that 47 per cent of their cases of asthma were nonsensitive to proteins. They present as a theory, in agreement with Alexander, that bronchial asthma should be regarded as something more than an effect resulting from a single process. They believe that there exists in asthmatic patients a constitutional imbalance of the vegetative nervous system in which an allergic factor may co-exist and be one of the many exciting agents of the paroxysm. They cite freely from the literature to show that experimental stimulation of the vagus in the neck produces bronchial constriction, bronchorrhea, cellular infiltration and emphysema. Thus they conclude that local infections of the respiratory tract may directly or indirectly irritate nerves supplying

the nasal and pulmonary passages, thus precipitating an asthmatic attack.

Out of a group of 409 cases of bronchial asthma in adults, 200 were found to be nonsensitive; a large percentage of other cases showed infection in the antra and ethmoids. In a smaller number of unresolved pneumonia there were many cases that were associated with sinus disease.

The authors have picked 19 cases, all of which had sinus involvement. They followed these cases for some three or four years, with conservative treatment, and marked improvement of the asthma was to be noted where the sinus condition had cleared up. It is the opinion of the authors that conservative nasal treatment yields fully as good, if not better results than nasal surgery.

Thomas E. Carmody, of Denver, in the October 3, 1931, issue of the *Journal A. M. A.*, offers an address on the publications of interest to the otolaryngologist, from 1925 to 1931. The achievements in our specialty of even five or six years are briefly stated and illustrations of Hasslinger's manikin are presented.

There is an editorial in the September 12, 1931, issue of the Journal, A. M. A., on Tonsillectomy which makes rather interesting reading to the otolaryngologist. Certain questions are asked about the physiology of the tonsil and the relationship of tonsil to adenoid and whether the removal of tonsils and adenoids at the same time is performing a maneuver in excess of the requirements of the case. In other words, should adenoidectomy be performed per se in indicated cases and tonsillectomy per se in cases where the post-nasal and nasal regions appear normal? A study is quoted from the University of Cincinnati College of Medicine, in which Selkirk and Mitchell have ventured to criticize many of the reports that have been published in the past. Their observations on children in Cincinnati are that three years after tonsillectomy and adenoidectomy there was a lessened incidence of colds, nasal obstruction and sore throats, while sinus infection, headache and growing pains were increased in frequency.

The interesting point brought out is that the indications and results are frequently judged by different persons, thus the laryngologist who performs the operation is less likely to observe conditions in a larger perspective such as the pediatrician or attending physician.

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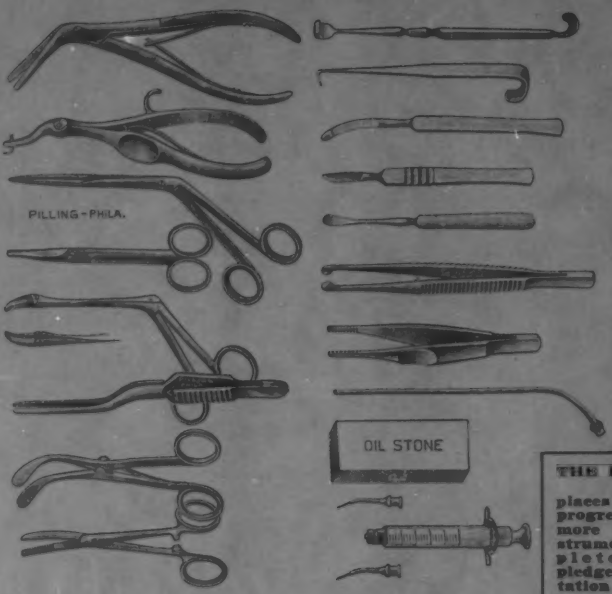

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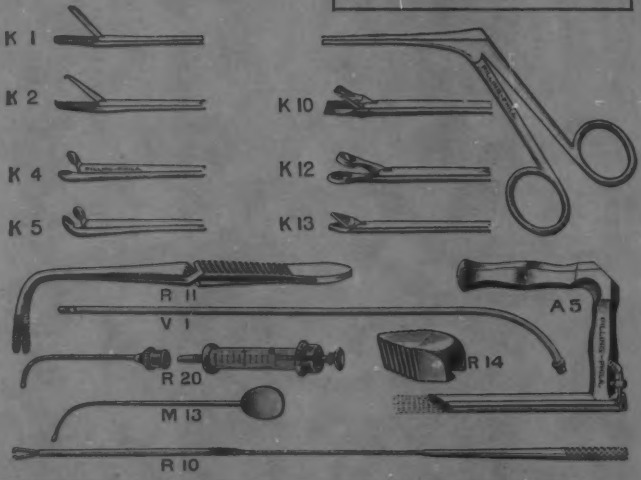

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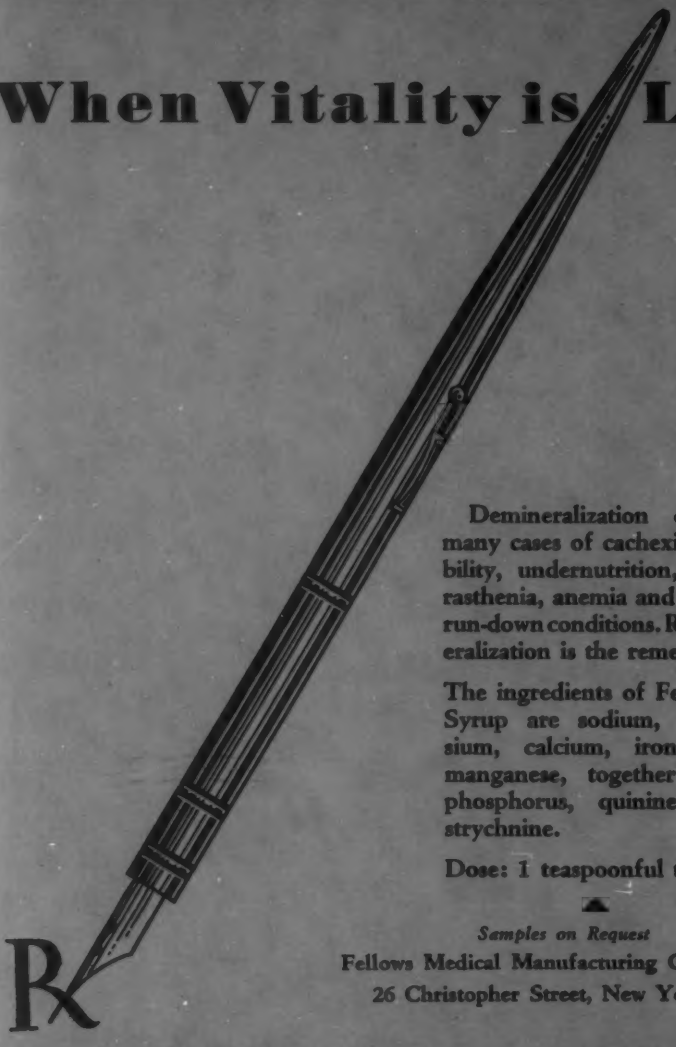

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